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THE SURGICAL PATHOLOGY OF BRAIN ABSCESS.[1]

By GEORGE SWINBURNE,
Melbourne.

THE majority of brain abscesses (over 80%) arise from an extension of some inflammatory focus in the ear or nasal accessory sinuses. A small percentage follow trauma to the skull, usually penetrating injuries, which involve the brain primarily, or secondarily by spread from osteomyelitis. Another small percentage are metastatic, occurring by vascular spread or embolism from an inflammatory focus elsewhere in the body, commonly from a lung lesion, often bronchiectasis or a lung abscess. Another less common primary lesion is osteomyelitis of a distant bone. Other infections sometimes associated with brain abscess are acute and chronic systemic infections such as endocarditis and septicæmia, prominent among the latter being those derived from carbuncle and furunculosis. Infections, acute and chronic, of the head and face apart from those affecting the accessory nasal sinuses form another group, and include infections in and about the orbit (some of these are associated with primary infections in the accessory nasal sinuses) and of the skin of the face.

Occasionally brain abscess follows acute tonsillitis, tooth extraction, infection by Vincent's organism and actinomycosis of the face.

The abscess which forms in brain tissue resembles in the main an abscess elsewhere in the body. There are certain minor differences consequent on the peculiarities in the structure, blood supply and absence of true lymphatics in the brain tissue. Tuberculomata and gummata are excluded from this description.

Infection may travel from an external focus outside the skull through the bone and meninges by the following pathways:

1. By direct extension (through rather than in the tissue planes):
   (*a*) Resulting from erosion of bone followed by necrosis of the *dura mater*.
   (*b*) Resulting from trauma.
2. By natural channels:
   (*a*) Blood vessels: (i) intravascular, inside the lumen of a vein or an artery; (ii) perivascular, via the space surrounding the pial artery or vein.
   (*b*) Perineural spaces (subarachnoid extensions): (i) nasal: surrounding olfactory nerves; (ii) aural: surrounding the eighth nerve in the internal auditory meatus; or via the sheath of the seventh nerve, thence to the subarachnoid sheath surrounding the seventh nerve in the internal auditory meatus.
   (*c*) *Vestibular channels: (i) via the aqueduct of the* cochlea; (ii) via the aqueduct of the vestibule.
   (*d*) Channels resulting from developmental anomalies: *(i) nasal: via a persistent cranio-pharyngeal canal;* (ii) aural: via persistent subarachnoid extension from the *hiatus subarcuatus* to the mastoid region; or via a persistent petro-squamous suture.

In the passage of infections to the brain a number of the above-mentioned routes may be used in sequence.

Atkinson[a] has pointed out that, as the central nervous system has no lymphatic vessels in the ordinary sense, and as there is no direct connexion between the intracranial contents and the general somatic lymphatic system, it is anatomically impossible for an infection to reach the intracranial contents by a lymphatic route. Turner and Reynolds[b] also were of the opinion that it seemed anatomically impossible for infection to reach the brain by way of the general somatic lymphatic system.

MacEwen,[c] on the basis of the results of injection experiments, stated that the lymphatics of the *dura mater* were abundant, lying in the spaces between the fibrous trabeculæ. These spaces were shown to be continuous

[1] The subject matter of this paper provided the material for two of a series of twelve post-graduate lecture-demonstrations in pathology, delivered in 1946-1947 under the auspices of the Melbourne Permanent Post-Graduate Committee to candidates for Part II of the Diploma of Laryngology and Otology in the University of Melbourne.

with the subdural space, and near the inner surface of the dura showed peculiar dilatations. They were also shown to be continuous with the dural veins. Hence, once this meshwork of capillaries within the dura was invaded by pathogenic organisms, the whole of the subdural space was open to invasion. It is possible that MacEwen's deductions were based on the results of faulty injection methods, as the above statement cannot easily be reconciled with the fact that infection is often held up by the dura clinically for long periods. The only known connexion between lymphatic channels and the veins is at the root of the neck.

Passage of Infection by Direct Extension Through rather than In Tissue Planes.

Erosion of Bone.

Infection by direct extension may result from erosion of bone followed by necrosis of the *dura mater*. Figure I is a diagrammatic representation of the cranial wall. In some parts it consists of compact bone and in others of an inner and outer plate with diploe in between. In the case of compact bone, erosion may gradually eat its way through the whole thickness of the bone to the dura, or at some stage it may open into an Haversian canal, and then pass by the blood vessels or lymphatic channels to the dural surface.

In diploic bone, the infection first goes through the outer table and reaches the diploe. It may then cause osteomyelitis with areas of necrosis and perforation of the inner table at some point, or may perforate the inner table more immediately, without wide spread in the diploe; or it may pass through by an infective thrombosis of one of the diploic veins.

The dura is resistant to a spreading infection and will hold it up for a time. The speed of passage of the infection will depend on the virulence of the organism concerned.

If the infection is held up by the dura, external pachymeningitis will occur. The dura becomes thickened and adherent to the bone; pus may form and collect between the dura and the bone, forming an extradural abscess. If this is not drained, or if the virulence of the organism is greater, the inflammatory reaction is likely to spread to the underlying arachnoid and *pia mater*, causing adhesion of these to the dura. This sealing process, when it has time to occur, is a protection against a general leptomeningeal spread; but if the process is progressive, the portion of the dura over the infected bone becomes softened and degenerated, and finally necrotic. The abscess is now in apposition with the *pia mater* and one of two things may happen.

First, the pia may also become softened and necrotic, and as a result of further spread, superficial ulceration of the subjacent cortex may occur, the brain tissue now forming part of the wall of the same abscess cavity, the opposite wall of which is formed by the carious bone. Naturally this abscess cavity may be irregular in shape and is usually collar-stud in type with an extradural loculus, communicating through a gap in the dura with an intradural loculus, the deep wall of which is composed of ulcerated cortex. This type of abscess is not uncommonly secondary to aural suppuration, especially over the *tegmen tympani* (or *tegmen antri*), and the fistula through the dura with the matted thickened meninges around it is often loosely described as a "stalk". This fact is mentioned because this type of brain "abscess" is frequently successfully drained and recorded as such, when in reality it is not a brain abscess but a cortical ulceration, the prognosis and treatment of which are vastly different from those of the true brain abscess. As the pus is drained with as little interference with the surrounding protective adhesions as possible, the patients recover, and no post-mortem evidence is available; some of these cases are almost certainly reported as of true brain abscess of the subcortical type. When a fistula in the dura is found at operation—and it is often tiny or even microscopic—this type of condition should be suspected.

Secondly, if the *pia mater* remains intact and the adhesions in the arachnoid mesh are not very firm, a subdural abscess may form, or if localization is not maintained, generalized leptomeningitis occurs. If the virulence of the organism is high, no time is available for sealing of the arachnoid mesh. Atkinson believes that in all cases of adjacent brain abscess a point of entry is to be found in the dura, and that if it is not found, it is because of an insufficient search, or more rarely, because the point is microscopic rather than macroscopic.

Of Atkinson's series of sixteen cases of brain abscess, a fistula through the dura was found in eight at operation; of 15 post-mortem specimens which he examined, and in which the brain adjacent to the focus of infection in the bone had not been disturbed, he found a macroscopic fistula in 15. Körner[4] found a path in 38 of 40 cases. Blau[5] collected 737 cases from the literature, and in only 40 was no fistula discovered. Carmichael, Kernohan and Adson,[6] on the other hand, in the examination of 75 brains with abscess, did not find a "stalk" macroscopically or microscopically in any instance. However, they did state that in many instances the dura was not available for study, and in others the "stalk" might have been obliterated by the process itself or by a subsequent operative procedure. MacEwen believes that a fistula is often present, but unfortunately gives no figures.

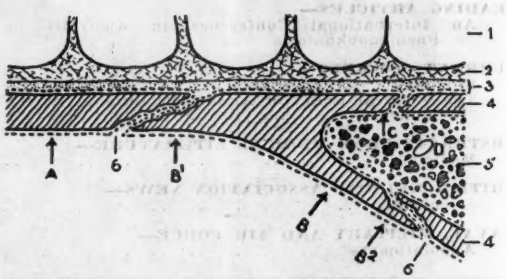

FIGURE I.

Diagrammatic representation of the cranial wall: 1, brain; 2, leptomeninges; 3, *dura mater*; 4, compact bone; 5, red bone marrow; 6, Haversian canal. (After A. L. Turner and F. E. Reynolds.[3])

When the spread has occurred by the venous route, and when it has occurred by mural thrombophlebitis (which may have resolved by the time operation or post-mortem examination is performed) rather than by retrograde thrombosis, then a definite fistula will not be found.

Trauma.

Infection resulting from trauma may be directly implanted in the cerebral tissue, or osteomyelitis, either directly consequent upon the trauma or resulting from a scalp infection caused by it, may be produced, further extension taking place either as described with regard to direct extension resulting from erosion of bone followed by necrosis of the *dura mater* (group 1 (*a*) in the tabulation), or by a sequence of routes, including some of those described below (group 2).

Passage of Infection by Natural Channels.

Blood Vessels.

The first type of passage of infection by natural channels is in association with the blood vessels. It may be intravascular (by the venous route or by the arterial route), or perivascular (via the space surrounding the pial artery or vein).

The cerebrum is supplied with arterial blood by two groups of vessels. The central branches pass from the circle of Willis at the base of the brain directly into its substance to supply the deep nuclei and the central part of the white matter. Coursing around the ventricle, these vessels then pass outwards towards the cortex, and branch and rebranch until they terminate as the so-called end arteries a short distance beneath the cortex. The cortical branches pursue a lateral course from the circle of Willis over the surface of the brain as the various cerebral arteries. They run in the subarachnoid

space. There are two types: (*a*) a large number of small vessels, which supply the grey matter only (*vide* Figures II and III), and (*b*) a smaller number of large vessels, which pass through the cortex to supply a narrow zone of white matter immediately beneath it. These latter also are so-called end arteries, and between their terminations and those of the central end arteries lies a narrow layer of white matter which has been for many years described as having no direct blood supply. This is the "avascular zone" stressed by Atkinson as being of importance in the pathogenesis of an abscess. The view that the terminal branches of both the central and the cortical arteries are end arteries is not now universally held. Pfeiffer[7] demonstrated the presence of a rich capillary anastomosis between the cortical and the central vascular systems of the brain. Lorente de No[8] maintained that the capillary network could be traced continuously from the olfactory bulb to the occipital cortex. Cobb[9] states that there are no such end arteries in the rabbit's brain.

However, as Carmichael *et alii* point out, it is obvious from these studies that some portions of the brain have relatively less vascular supply than others, and that

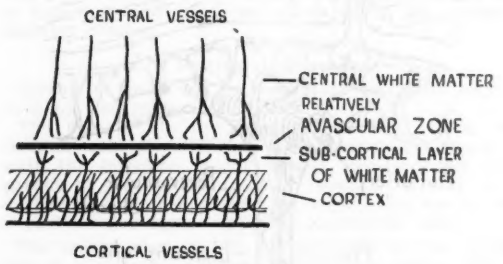

FIGURE II.

Diagram of relatively avascular zone in cerebrum. (Modified from E. M. Atkinson.[10])

Atkinson's avascular zone can be described as relatively avascular; but just what relation this fact has to the site of predilection for abscess is difficult to estimate.

In the cerebellum there is a similar arrangement of the vessels, except that owing to the greater convolution of the cerebellum a folding of the avascular layer upon itself occurs in the core of the folium (*vide* Figure III). The vessels lie in the fissures in the subarachnoid space, and their branches pass through the cortex in the same way as in the cerebrum, some supplying the cortex and the remainder passing through to the white matter. The veins lie with the arteries in the fissures and often partially surround them, as veins commonly do in other parts of the body. At the surface they leave the artery to join a venous sinus, but each vein drains the area supplied by its accompanying artery.

Perivascular Route.

The perivascular spread of infection may be via the perivascular space surrounding a pial artery or vein. Figure IV is Weed's well known diagram showing the perivascular space or sheath and its relation to the cerebro-spinal fluid circulation.

Atkinson, in 16 specimens examined microscopically, found 13 in which this path was that taken by the infection. He concluded that this was the commonest mode of spread into the brain itself. The infection must have reached the subarachnoid space before this pathway becomes available.

Figure V shows the first stage in the spread of an infection by the perivascular route in a cerebellar abscess in Atkinson's series. The pial vessels are patent, but their surrounding perivascular sheath is distended by gross infiltration with inflammatory cells; these are usually mainly mononuclear. The perivascular sheath is adherent to the cortex of the brain; in some places the whole is converted into granulation tissue with no visible line of demarcation. This perivascular infiltration and adherence to the cortex continue down into the fissure. The appearances are the same in a cerebral abscess. The spread against the normal flow of cerebro-spinal fluid can be explained by the fact that adhesions surrounding the area prevent extension through to the main subarachnoid space, and upset the local pressure relations of the cerebro-spinal fluid, so interfering with its normal direction of flow. The localized nature of the attack upon the brain is well shown in Figure V, in which the infiltrated perivascular sheath is adherent to one lobule which shows gross pathological changes, while the neighbouring lobule is comparatively normal.

In the affected cerebellar lobule there is an increase in the depth of the whole cortex due to œdema, both molecular and granular layers being involved; there is considerable new vessel formation, the cortex being

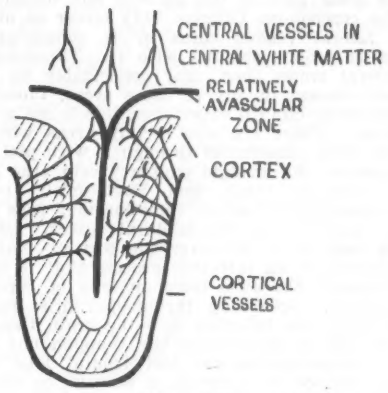

FIGURE III.

Diagram of relatively avascular zone in cerebellum. (Modified from E. M. Atkinson.[10])

broken up by large capillaries which pass inwards from the pial vessels. Under high power magnification the greatest change is seen in the granular layer, where the nuclei tend to be arranged in rows or clumps as a result of the œdema, and the whole—but especially this layer—is invaded by inflammatory cells radiating from the vessels, by large numbers of endothelial cells, and by some large phagocytes. Often, particularly in the more chronic abscesses, there is some glial hyperplasia (see Figure XIV).. In the parts most affected the Purkinje cells are absent; further away from the centre of infection they are sometimes displaced deep into the molecular layer, their outline becomes blurred and they stain poorly, their nuclei begin to disappear and they lose their tendrils. All these are manifestations of degeneration. Brunner[11] in 1926 described almost identical appearances in his sections.

A deeper section in the same case (see Figure VI) shows how the abscess begins in the white core of the folium at the site of the relatively avascular zone described by Atkinson. From its point of origin in the centre of the lobule the abscess tends to spread inwards along this relatively avascular zone until it meets and is stopped by the central mass of white matter with its relatively good blood supply. At the same time the abscess extends backwards or along the lobule, again in the relatively avascular zone, and until a comparatively late stage it usually remains confined to one lobule because of the vascularity of the cortex around it and of the central white matter deep to it. It is difficult to obtain evidence of a similar spread within the brain in the case of a cerebral abscess, because it extends from its origin in all directions and so destroys its site of origin. However, in nine out of eleven cases of cerebral abscess Atkinson found undeniable evidence of a perivascular route of infection, and as he states, it is reasonable to presume from our knowledge of the anatomy of the cortical cerebral vessels that the same conditions

would obtain and that the abscess would start in the relatively avascular zone which is situated in the white matter a short distance deep to the cortex.

Carmichael *et alii* maintain that in perivasculitis associated with brain abscess it is difficult to distinguish cause and effect, and they have found the same type of perivasculitis repeatedly in connexion with abscesses of known metastatic origin. However, they do not doubt that this mode of transmission does exist in a certain number of cases.

Intravascular Route.

In the "intravascular" group the infection may spread by veins or arteries.

Venous Spread.—When the infection spreads via the veins it takes the form of a retrograde thrombophlebitis. The venous drainage of most of the temporo-sphenoidal lobe, of a great part of the parietal and occipital lobes and of the cerebellum (Figure VII) either is ultimately into the lateral venous sinus or is connected by an anastomosing venous channel with it. Thrombophlebitis of the lateral sinus, then, may conceivably be followed by a brain abscess in any of these areas, though, as is well known from clinical experience, this is not a common complication. There are also certain areas where veins outside the dura anastomose through it with pial veins,[12] as, for example, the mastoid emissary vein (the posterior temporal diploetic vein), and Eagleton's[13] points of "venous vulnerability", where infection may pass from the extradural tissues into the subdural spaces. Lastly, the wall of a vein, as it lies over the tegmen, may happen to be involved in an infective process that is spreading directly through the dura from an area of carious bone in this region. Spreading thrombophlebitis may supervene and carry the infection into the brain tissue. This method of spread is probably the common one in those cases of temporo-sphenoidal and frontal lobe abscesses which are formed as a result of passage by the venous route, and spread via the lateral sinus is more common in the case of cerebellar abscesses. The histological appearances are best seen in the cerebellum, but are the same in cases of temporo-sphenoidal abscess. Figure VIII is a photomicrograph from one of Atkinson's cases of cerebellar abscess, actually the only case of cerebellar abscess with a retrograde thrombotic route in his 1926 series of sixteen brain abscesses examined microscopically. A large pial vein is shown containing breaking down clot, with a few smaller veins also thrombosed surrounding it. The artery and its branches are patent. Deeper in the fissure, at the entrance to which these vessels lie, the same appearances are seen in an artery and vein there, and perivasculitis is seen around both sets of vessels, but much less in degree than that which occurs when infection occurs by the perivascular route. Perivasculitis is present because the clot in the vein is infected; the cells are mostly polymorphonuclear, whereas they are mainly mononuclear in type in the perivasculitis associated with the perivascular route. It is to be noted also that the perivascular sheath has not merged with the cortex which shows little change either on its surface or in its substance.

As the infection extends, the area of the brain, cerebrum or cerebellum drained by the veins emptying into the thrombosed pial vessel or vessels starts to undergo degeneration because of interference with its blood supply; in the layers of the cortex the number of cells has diminished, their nuclei stain badly or have disappeared, and in the case of the cerebellum the Purkinje cells begin to alter in shape and staining. Here again the degenerative changes are most evident and begin first in the relatively avascular zone. In Figure IX an abscess is shown developing in the white core of the lobule, the cortex being respected by the suppurative process for the most part. However, extension follows the course of the thrombosis outwards towards the surface, and the surface cortex is the earliest to be destroyed; this is beginning to occur in Figure IX. Multiple abscesses involving more than one lobule are more common in this group, especially if the pial thrombosis is the result of thrombosis of one of the large venous sinuses.

Arterial Spread.—Unless by the internal carotid, the vertebral, the middle meningeal or the accessory meningeal artery, infection cannot pass from outside the dura into the brain via an artery. On the other hand, infection can be carried by a pial artery which lies over the tegmen and has become involved in an infective process that has gradually spread through the dura. Conceivably, in these circumstances, a mural thrombus may form and become dislodged, or a clump of bacteria may be swept off the thrombus, carrying infection into the white matter via one of the terminal end arteries, and cause the formation of an abscess; or the whole lumen and branches beyond the point affected may be occluded by thrombosis. In each case an area of infarction in the brain is produced, more or less extensive, which becomes infected. Because this infarction occurs suddenly, it is accompanied by a sudden onset of symptoms, which may take the form of a convulsive seizure and be followed by a varying degree of paralysis or loss of sensation, the severity

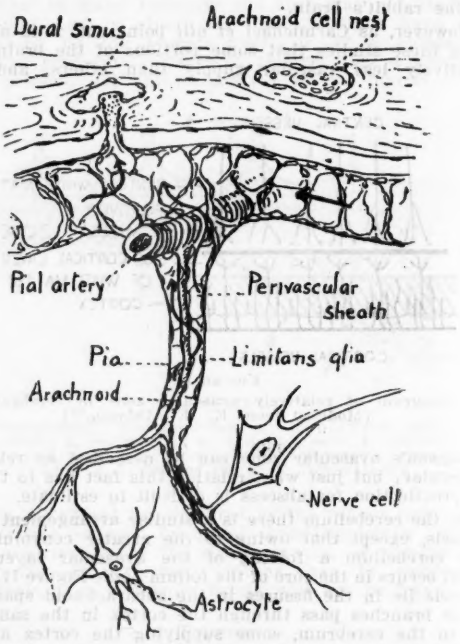

FIGURE IV.

Diagram to show perivascular sheath and cerebrospinal fluid circulation. (After Weed.[10])

depending on the size of the area of brain affected and its site. Strictly, such cases, except those in which the vessel is completely thrombosed, are of metastatic origin. The appearances of the brain are different from those seen with other methods of spread, the cortex around the thrombosed vessels shows very little change until the area of the abscess is reached; even here the surrounding white matter is abnormal only close up to the abscess cavity, where a narrow band of tissue showing hæmorrhage, thrombosis and some inflammatory infiltration, is encountered. Outside this area, the veins are slightly dilated, but there is not the venous distension and areas of hæmorrhage which are found in the abscesses formed by retrograde thrombophlebitis. These appearances are those one would expect to find around an infarct where the tissues have had time to become depleted of blood by the veins; in venous thrombosis, the tissues are engorged with patches of hæmorrhage, because the venous outlet is cut off while the arterial inlet is still patent.

Perineural Spaces (Subarachnoid Extensions).

Nasal infection can pass from the nasal cavity into the subarachnoid space via the perineural sheaths around the

olfactory nerves, which are prolongations of the pia-arachnoid space accompanying the nerves through the cribriform plate into the olfactory epithelium of the upper sixth of the nasal cavity.

Aural infection can spread via the perineural space surrounding (*a*) the eighth or (*b*) the seventh nerve in the internal auditory meatus.

In the first instance the infection would have passed from the middle ear or mastoid antrum to the internal auditory labyrinth, then from the cochlea to the sheath of the eighth nerve and into the internal auditory meatus. Rarely infection may become localized in the internal auditory canal; but usually it passes on to the general leptomeningeal space, producing meningitis. This meningitis may be further localized for a time in the lateral cistern of the posterior fossa (see Figure X).

In the second instance infection involves the facial nerve sheath somewhere on its course, usually while it is in the Fallopian aqueduct, or in its passage over the labyrinth, and passes along it into the internal auditory canal. Friesner and Braun[14] mention this as a possible path, and Atkinson had a case in his series. He points out that the route should be kept in mind in cases of facial paresis following chronic suppurative *otitis media* or following operation, in which signs of intracranial infection develop.

Vestibular Channels.

Spread of infection by vestibular channels may take place by two routes.

The first is via the aqueduct of the cochlea, which communicates directly with the subarachnoid space. Here again the infection is not usually localized, but quickly spreads to produce meningitis. However, it may on rare occasions be walled off and be responsible for a venous or perivascular spread into the cerebellum later.

The second route is via the aqueduct of the vestibule. This is a narrow canal with a smaller lumen than the aqueduct of the cochlea. This is probably the commonest route of infection from the labyrinth to the cerebellum. There are two variations in the method of spread: (*a*) erosion of the bony wall of the aqueduct from outside may occur, with the production of an extradural abscess; (*b*) empyema of the saccus may rarely occur (one case in 35 after labyrinthitis, according to Fraser[15]), following spread of infection along the *ductus endolymphaticus* which lies in the vestibular aqueduct, causing an abscess in its blind end. Further spread to the brain in either case is either direct, or via the subdural vessels or their perivascular spaces (see Figure X).

Channels Resulting from Developmental Anomalies.

Channels resulting from developmental abnormalities may be nasal or aural.

Nasal spread may occur via a persistent craniopharyngeal canal from the post-nasal space. Atkinson mentions this route as a possibility and admits its great rarity.

Aural spread may occur, firstly, via the *hiatus subarcuatus*. The dura and arachnoid dip into the subarcuate fossa, and in some cases the prolongation of the subarachnoid space passes deeply toward the mastoid antrum, which it reached in fœtal life. This must be a rare route; but infection of the bone around the mastoid antrum may involve such a persistent extension from the subarcuate fossa (see Figure X).

Aural spread may also occur via a persistent petro-squamous suture. This suture sometimes persists in a child, and infection may extend from the middle ear cleft via this suture to the middle fossa dura, forming an extradural abscess.

The Further Development of a Formed Brain Abscess.

Owing to variations in the blood supply to parts of the brain, and depending on the path the infection takes, whether perivascular or intravascular, the extension of infection and the further development of an abscess after its early stages of formation differ a little in each case, and also in the cerebrum as opposed to the cerebellum.

In the cerebrum the grey matter of the cortex, probably because of its good blood supply, offers considerable resistance to the outward extension of an abscess which has begun in the relatively avascular zone; but the infection spreads fairly rapidly away from the cortex toward the ventricle, in a general direction parallel with the course of the vessels, and the spread takes place in this direction mainly because tissue destruction is consequent upon thrombosis of the vessels around the edge of the abscess. Perforation of the ventricle will ultimately occur, though a valve-like leak may precede a complete rupture for a time. Spreading leptomeningitis follows, and because of the flood of organisms is usually fatal. With modern chemotherapy the mortality may be reduced; a sufficiently large number of cases has not yet been recorded to allow of any conclusions to be made on this point.

Atkinson, quoting Miodowski[16] and Urbantschitsch, states that in chronic cases with a slow advance the chorioid plexus may be found adherent to the ventricular wall, reinforcing it where the abscess is nearing it and so delaying perforation for a time.

In a long-standing case most of the white matter of a convolution, or even of a lobe, may be destroyed, a shell of cortex which becomes progressively thinner being left. Sometimes the abscess bursts through the cortex and drains via the original pathway through the adherent pia and dura and bone, into the mastoid or frontal sinus —an evacuation along the "stalk". This was commoner in MacEwen's time than today, because nowadays treatment is begun at an earlier stage. MacEwen called this "*otorrhœa cerebralis*", and it is characterized by a great increase in the discharge of pus from the ear, with temporary relief of the symptoms caused by the abscess. A "stalk" is not nearly so commonly present in cerebellar abscesses, and evacuation via such a "stalk" is extremely uncommon. Although it is more usual for an abscess to perforate into the ventricles, it may on rare occasions burst or leak into the subarachnoid space. Loculation may occur as a result of a fresh abscess forming in the wall of the original abscess, or there may be two or even three of these smaller extensions around the parent abscess. Usually they have a small, narrow connexion with the main cavity, but they may not, being then separated by a wall of inflamed tissue, so that the appearance is that of two separate abscesses of different ages. The shape of a cerebral abscess is often like that of a pear with the broader base toward the ventricle.

In the cerebellum the extension is governed by the same factors, but varied by the increased cortical convolution and by the difference in the vessels consequent upon this, and by the absence of ventricles. Extension occurs, as we have seen, at the expense of the white matter of one lobule at first, and in fact, although the adjacent lobules may become inflamed, the active suppuration rarely extends beyond the one lobule.[13]

As in the cerebrum, the grey matter of the cerebellar cortex offers resistance, and although it is pushed away to either side of the growing abscess and becomes swollen and distorted with œdema and inflammation, it is not destroyed until late. Death usually occurs from medullary pressure before the cortex gives way; but this does happen, and not uncommonly in the form of a leak.

The shape of a chronic localized cerebellar abscess is usually ovoid, with regular smooth walls. The lateral extensions seen in the cerebrum occur in acute cases, but then only into the central white matter. They increase the difficulties of drainage. Multiple abscesses sometimes follow lateral sinus thrombosis, as a result of a retrograde thrombosis of two or more vessels at once. This condition should be suspected in a case of abscess following lateral sinus thrombosis if progress is not favourable after drainage of one abscess.

Clinical Classification of Brain Abscesses.

Brain abscesses, which include cerebral and cerebellar abscesses, may be classified clinically as (i) adjacent and (ii) non-adjacent.

The adjacent brain abscess group includes all those brain abscesses which occur in the immediate neighbour-

hood of a focus of infection in the bones of the skull, and may be subdivided into the following subgroups: (*a*) otogenous, (*b*) rhinogenous, (*c*) osteomyelitic and (*d*) traumatic.

Into the non-adjacent brain abscess group fall the metastatic abscesses arising by blood-borne infection from a distant focus of infection. This does not include those abscesses which make use of a hæmatogenous path, along with a sequence of others, in passing from an adjacent focus to the brain in its immediate neighbourhood.

Adjacent Brain Abscesses.

Adjacent brain abscesses are usually single; they may occasionally become large, even involving the whole of one and part of a neighbouring lobe. Sometimes two separate and distinct abscesses are found, especially in the cerebellum, in association with a thrombosed lateral sinus.

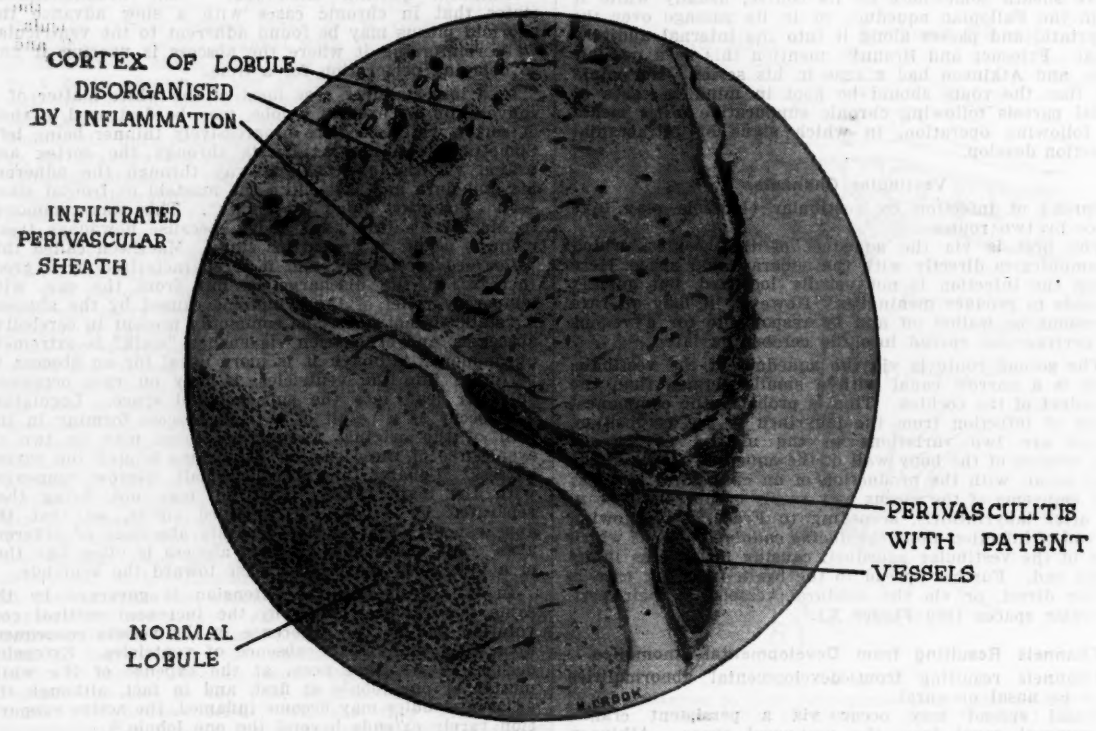

FIGURE V.

Marked perivasculitis with inflammatory process attacking one lobule only. (× 200; modified from E. M. Atkinson.[9])

Less commonly they are multiple. These multiple abscesses sometimes take the form of connected loculi, or are found as one large abscess with several small abscesses surrounding it, which are not truly separate multiple abscesses, but result from extension of infection from the large abscess.

Adjacent brain abscesses are, as a rule, secondary and adjacent to a focus of infection in the ear, in the mastoid process, in the closely related lateral cerebral venous sinus, or in the nasal accessory sinuses (usually the frontal sinus), and they are usually found in the frontal or temporo-sphenoidal lobes or in the cerebellum. The infection passes from the focus to the brain by one or a combination of the paths described in a previous paragraph.

Rarely an abscess is found in the parietal or occipital lobe, or even in the base of the brain or brain stem. These rarer examples may result from vascular spread from an otogenous focus of infection as a result of retrograde venous thrombosis spreading from a thrombosed lateral venous sinus, or from one of the venous sinuses which lie in association with the petrous pyramid. More commonly they result from a localized area of osteomyelitis of the skull situated over the particular lobe affected, and they are adjacent to the osteomyelitis. Sometimes the focus is a spreading osteomyelitis which has extended from an otogenous or rhinogenous focus, especially the latter.

Traumatic Brain Abscess.

Traumatic brain abscess may follow punctures or penetrating wounds of the brain, resulting usually from the direct implantation of an infected foreign body, gunshot wounds and bomb fragments being a specially common cause during times of war. Prevention lies in correct treatment of the wound at the time. Punctured or penetrating wounds of the scalp or skull producing osteomyelitis may, of course, be followed by an adjacent brain abscess.

Traumatic brain abscess may also follow compound fissured fractures of the skull, or fractures of the base involving the *tegmen tympani* (or *antri*), or the cribriform plate of the ethmoid, where there is an accompanying tear in the dura. These are also adjacent brain abscesses with a traumatic gap as a path instead of a pathological path. Abscesses following operative procedures in the nasal or aural regions are also of this nature, some injury to the dura having taken place. Intact dura has a strong resistance to the passage of infection.

Foreign bodies may lie dormant in the brain for weeks, months or years without symptoms, then as a result of a blow on the head, a too early plastic procedure to the cranium or some other cause, a flare-up may occur and an abscess may develop around the foreign body. Rarely a hæmatoma formed in the brain as a result of *contre-coup* may become infected via the blood-stream and form an abscess.

Traumatic abscesses due to direct implantation vary in size; they may be superficial and be shaped in a flattened ovoid fashion, or be two to two and a half inches deep and elongated, as King[13] describes it, "like an old-

fashioned money bag". They may also be irregular, and sometimes there are pockets or extensions in the shape of smaller surrounding abscesses.

Most observers agree that up to 90% of these abscesses are single. The cavity sometimes has a fistulous connexion with the outside of the cranium and sometimes contains air. In former times, when tubes were wont to be left in after gunshot wounds, a thick, sometimes quarter-inch wall would often develop around the tube, and though it would collapse partially on removal of the tube, it would harbour latent infection. In the recent war, interference of this nature was happily uncommon.

Non-Adjacent or Metastatic Brain Abscess.

Metastatic brain abscesses are usually situated in the white matter, away from the site of the primary infection. They have an apoplectic onset. The organisms are carried by the blood-stream from the distant primary focus and may occur in the course of any pyæmia.

The clinical types fall into the following two groups.

1. The first group comprises abscesses occurring in the course of an overwhelming or progressive generalized septic process. These are usually multiple, and the patient is so ill that the cerebral symptoms are attributed to the acuteness of the toxæmia. Even if abscess is suspected, localization is difficult, and operation is contraindicated because of the condition of the patient and the probability of multiple abscesses.

2. The second group comprises abscesses secondary to subacute or chronic infections in the body, which can be subdivided into (i) pulmonary and (ii) extrapulmonary infections.

Pulmonary or pleural suppuration is the commonest cause of metastatic brain abscess; the lesions include (*a*) bronchiectasis, (*b*) empyema and (*c*) lung abscess. It is not definitely known why lung suppuration should so commonly cause secondary brain abscess, and why such a complication is relatively rare in ordinary pyæmic states. Carcinomatous secondary deposits in the brain are also common when the primary deposit is in the lung, but rarer when the primary deposit is elsewhere.

A number of earlier observers thought that some of these cases were probably not metastatic at all, but that both the brain abscess and the lung disease were secondary to paranasal sinusitis. There is certainly a high incidence of paranasal sinusitis in cases of bronchiectasis and of pneumonic conditions leading to empyema; Collis[19] went into this matter carefully and published his results in 1944. He gathered some evidence that infected material was carried to the brain from the chest via the spinal veins. He thought that this was probably also the path of secondary deposits to the brain in carcinoma of the lung and breast. He gave reasons why the arterial embolic hypothesis, the concomitant paranasal sinus infection hypothesis, and the hypothesis which presupposed that these abscesses were produced by spirochætes or by a virus or another organism not easily identifiable, were unlikely to be valid.

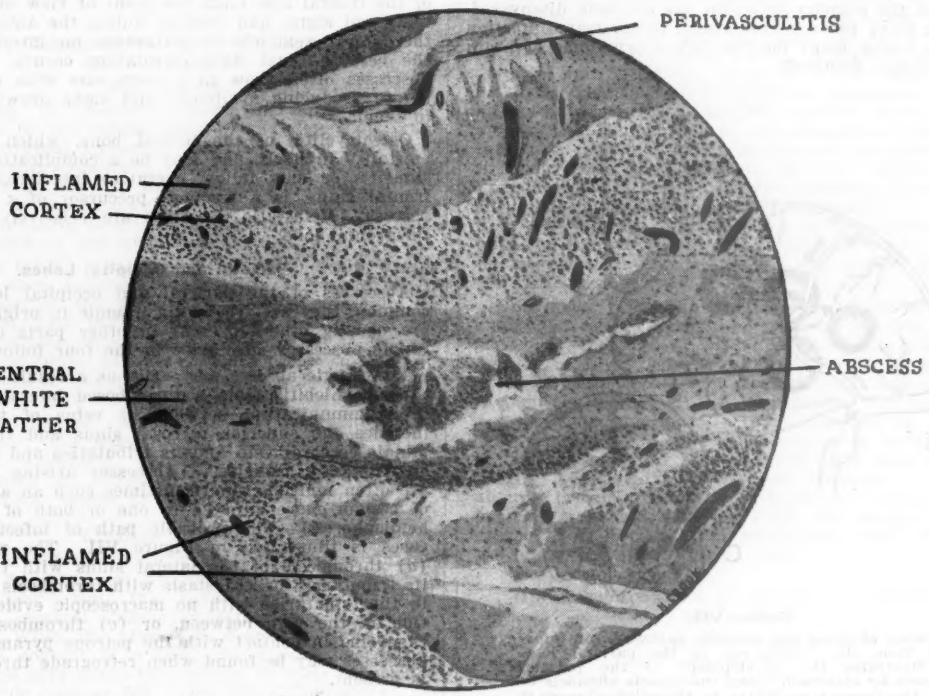

FIGURE VI.
Abscess in centre of white matter of a lobule. (× 200; modified from E. M. Atkinson.[2])

He thought that the spinal vein system, which is valveless, under certain conditions, as in coughing or straining at stool or lying flat on the back, could flow in a cerebral direction, and an infected embolus originally coming from a venous mural thrombus could pass to the cerebral venous circulation. The spread from the lung to the chest wall (and hence to the intercostal veins and the azygos and so to the spinal veins) occurs through vascularized adhesions in the pleural cavity, which accounts for the fact that the lung lesion must be long-standing, as is the case in bronchiectasis and lung abscess. In the case of empyema, the chest wall is already adjacent. Operative interference in any of the three conditions producing an infected sinus close to an intercostal vein and its tributaries will increase the risk. The bronchial veins are also tributaries of the azygos, and any lesion of their drainage area adjacent to the main bronchi, including carcinoma, can metastatize via

the spinal venous bed. Having reached the brain, the thrombus will become lodged at the venous end of the capillary bed.

King, after reviewing the literature, found that 50% of metastatic brain abscesses were single. Parker[30] found this figure also in the Mayo Clinic series. They can often be localized, but the prognosis is usually poor, because the patients are often already debilitated from long-standing pulmonary suppuration.

Of the extrapulmonary infections causing brain abscesses, examples are osteomyelitis, renal sepsis, cystopyelitis (in these last two the organisms may be *Bacillus coli communis* or some other coliform organism), rarely endocarditis, or infection from the tonsil. Metastatic abscesses may, of course, be otogenous or rhinogenous in origin, though this is uncommon. An abscess may even follow dental extraction for an apical abscess. The occurrence of a so-called idiopathic brain abscess simply means that the primary infection has not been discovered. Indeed, in many cases in this group, the primary infection may have healed many months before symptoms of brain abscess become manifest.

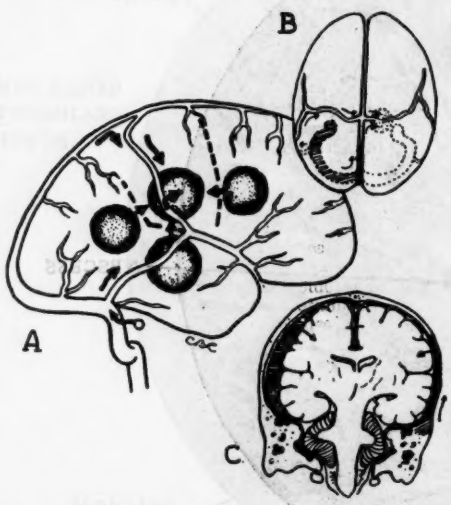

FIGURE VII.

Drawing showing the possible pathways of infection from the middle ear to the parietal lobe. A illustrates the development of the parietal abscess by extension along the venous channels or the development secondary to the otitic abscess in the other lobes; B, an insert, shows the possible pathways of a parietal abscess in the hemisphere opposite the draining ear, although such an abscess may be due to an apparently unaffected ear on the same side; C, an insert, shows the situations which may theoretically result in a parietal abscess —a subdural abscess on the left and osteomyelitis of the temporal and the parietal bones on the right. (After Courville and Neilsen.[23])

REGIONAL PATHOLOGY.

The Temporal Lobe.

Adjacent temporal lobe abscess is secondary to aural infection, and the infection may traverse one of the following paths. (i) It most commonly passes upwards through the roof of the attic or mastoid antrum to the middle fossa and thence into the temporal lobe through its inferior surface. (ii) More rarely, infection may pass through the perilabyrinthine cells to the cells beneath the antero-superior surface of the petrous portion of the temporal bone, and so into the temporal lobe, either directly, or following involvement of the superior petrosal sinus. (iii) The superior petrosal sinus may be affected by a retrograde infection from the lateral sinus, although abscess of the posterior fossa more commonly follows infective thrombosis of the lateral sinus. In this connexion it is well to remember that one of the main venous efferents of the basal portion of the brain, one of the medio-cerebral veins, empties into the superior petrosal sinus.

The Frontal Lobe.

Frontal lobe abscess is rarer than temporo-sphenoidal or cerebellar abscess, and usually follows chronic, sometimes acute, suppuration in the frontal sinus, or less commonly in the ethmoid labyrinth. The paths of infection are similar to those in otogenous cases.

Because of the less rich blood supply of the frontal lobe, abscesses in this region tend to extend and to grow more quickly than in other lobes. For the same reason there is a poorer reaction to infection, and operative trauma is withstood less well, with a higher relative operative mortality. Because the anterior horn of the lateral ventricle passes well forward into the lobe, its perforation is not uncommon. But because of the "silent" nature of the frontal lobe from the point of view of focal symptoms and signs, and because unless the anterior horn of the lateral ventricle is perforated no interference with the cerebro-spinal fluid circulation occurs, frontal lobe abscesses often grow to a large size with a thick wall before producing symptoms and signs drawing attention to them.

Osteomyelitis of the frontal bone, which has a high mortality in itself, and may be a complication of frontal sinusitis or may follow operative interference with the frontal sinus, is a frequent precursor of a frontal lobe abscess. Metastatic abscesses are relatively common in the frontal lobe.

Parietal and Occipital Lobes.

Abscesses of the parietal and occipital lobes are uncommon, they are frequently pyæmic in origin, and there are usually others present in other parts of the brain. Single abscesses may arise in the four following ways.

1. A single abscess may arise as a result of retrograde thrombophlebitis from a thrombosed lateral sinus. The intercommunicating network of veins of this region[23] includes the superior petrosal sinus and the superficial middle cerebral vein and its tributaries and their smaller anastomosing radicles. (Abscesses arising in this way are often multiple, and sometimes such an abscess is one of two or more situated in one or both of the cerebral hemispheres.) The probable path of infection in these cases is illustrated in Figure VII. The mechanism is (*a*) thrombosis of the lateral sinus with thrombosis in its tributaries, or (*b*) stasis with thrombosis further back in the tributaries with no macroscopic evidence of infection in the vein between, or (*c*) thrombosis in one of the veins in contact with the petrous pyramid. Multiple abscesses may be found when retrograde thrombophlebitis is present.

2. A single abscess may arise from a focus of infection in the overlying scalp or bone. The bony osteomyelitis may be localized; it is generally a result of trauma, or it may have spread upwards from an aural infection in the same manner as frontal osteomyelitis spreads.

3. A single abscess may be secondary to an otogenous subdural abscess, usually due to low-grade infection and walled off for a time.

4. A single abscess may arise through a spread of suppuration from the temporal lobe. In this connexion it must be remembered that, to reach the parietal lobe from the temporal lobe, the Sylvian fissure must be crossed by the help of adhesions, with the formation of a fistulous track. The path by the external capsule is very unlikely.

Parietal abscesses may rupture into the lateral ventricle, either through the fibres of the *corona radiata* to burst into the lateral margin of the body of the ventricle, or through the *corpus callosum* to break through its roof.

Cerebellum.

There are certain sites on the surface of the cerebellum where infection most commonly enters. Adjacent abscesses are almost all found in either the postero-inferior or postero-superior lobules, the reason being that most of

the paths by which infection reaches the cerebellum either open in the vicinity of, or lie close to, these two lobules or the great horizontal fissure which lies between them (see Figure X)—namely, the passages from the labyrinth, the lateral sinus and the area of bone lying between the two. Because all these except the *hiatus subarcuatus* lie in close association with the postero-inferior lobule, this lobule is the most commonly affected, and the infection enters by the great horizontal fissure or the one below it.

If the infection travels by the *hiatus subarcuatus* or from an area of carious bone high up on the posterior petrous wall, the postero-superior lobule will be affected. This type of abscess is practically always confined to the one lobule.

Abscess of the brain secondary to infection of the sphenoid sinus is comparatively rare. Lischoff,[33] in reporting a case in 1935, could find only five cases in the literature. Cavernous sinus thrombosis is a more common complication.

The Cerebro-Spinal Fluid Changes in Brain Abscesses.

Cell Content.

The passage of infection through the dura is usually accompanied by the sudden onset of the signs and symptoms of meningeal irritation, usually a rigor and headache, sometimes vomiting. A shower of polymorphonuclear cells appears in the cerebro-spinal fluid at the same time. If the subarachnoid space has escaped with a localized invasion producing mild symptoms, this stage may be missed in the history altogether.

After the initial invasion of the brain itself, during the period when localized encephalitis is proceeding to abscess formation, the proportion of lymphocytes in the cerebro-spinal fluid increases relatively to that of the polymorphonuclear cells. As a general rule it may be said that the better the encapsulation, the smaller the number of cells in the spinal fluid and the less in number the neutrophile polymorphonuclear cells.

The persistence or reappearance of neutrophile polymorphonuclear cells suggests that encapsulation is not progressing favourably. A definite increase may be associated with an extension of the abscess, involving new perivascular spaces or approaching a ventricle.

Chloride Content.

The normal chloride content of cerebro-spinal fluid is 725 to 750 milligrammes per 100 millilitres. Greenfield and Carmichael[39] state that a reading below 700 milligrammes in cerebral abscess indicates generalized leptomeningitis. When the abscess is deep and encapsulated the chloride content is normal, the reading decreasing as the abscess approaches the surface. In their experience, if the chloride content fell below 680 milligrammes per 100 millilitres, the patient died.

From these findings it is deduced that the greater the meningeal involvement, the lower is the chloride content—that is, the latter varies inversely with the cell count. Atkinson found this to be so in a limited series of cases.

Protein Content.

The protein content of cerebro-spinal fluid consists of a trace of serum-globulin and of albumose, the total albumin content being from 25 to 80 milligrammes per 100 millilitres (average 35 milligrammes).

Increase of albumin with increase in the cell content is found in all forms of meningitis, in cerebral abscess and tumours, in epidemic encephalitis and in poliomyelitis, and the two phenomena go hand in hand in these diseases; hence the determination of protein content is of little help in brain abscess.

Isolated increase of protein content without any cellular increase—the so-called "*dissociation albumino-cytologique*"—occurs in brain and spinal cord tumours, and also in cases of cerebral arteriosclerosis, in which cerebral thrombosis or capillary hæmorrhage has occurred in the neighbourhood of the ventricles and basal cisterns (Greenfield[47] and Carmichael, 1935).

Bacteriology.

The reports of investigators on the bacteriology of brain abscess have been conflicting, and there is no real unanimity of opinion on the pathogenicity of the various organisms commonly found. This lack of definite knowledge on the subject appears to be due to the omission of adequate bacteriological investigation of each case, or to careless and incomplete investigation. Until a complete and methodical investigation of the pus in every case, including anaerobic cultural examination, is carried out, the present uncertainty will continue. Typing of streptococci and pneumococci should be carried out.

According to the majority of observers who have recorded series of cases, the streptococci are the commonest organisms found in brain abscesses, then pneumococci, which are not infrequently mixed with staphylococci. Other organisms found by Bagley[34] are *Bacillus proteus*, *Bacillus coli communis* and atypical coliform organisms, diphtheroids, anaerobic organisms including *Bacillus fusiformis* and *Clostridium welchii*; odd cases have been recorded as due to actinomycosis, and there have been several cases of abscess due to *Streptococcus viridans* (Mendel and Saibel[35]). There is a difference of opinion as to the virulence of the different organisms and hence as to the prognosis. Lund,[30] from his series, is of the opinion that primary aural and rhinogenous *Bacillus coli* infections tend to produce intracranial complications, and in such cases the organism is very virulent. It is associated with such chronic primary infections as chronic osteitis or cholesteatoma, because it requires a diminished local resistance for the development of its pyogenic characteristics. In its presence alone the tissues do not form a good barrier, and perforation of the ventricle is common.

Not much is known of the importance of the true anaerobes in the formation of brain abscess, and this aspect urgently needs investigation. The consensus of opinion of investigators in general seems to be that the presence of anaerobic bacteria in a brain abscess does not favour the formation of a capsule, whereas that of aerobic bacteria, especially the cocci, does so. Staphylococci, streptococci and pneumococci seem equally capable of forming capsules. Mixed aerobic and anaerobic organisms as a rule do not form good capsules. A walled-off abscess is not necessarily a quiescent abscess—virulent bacteria may be present within the cavity, and their escape may produce fatal meningitis; only occasionally is the content of an abscess sterile.

Histopathology of the Abscess and Surrounding Brain Tissue.

The study of the earliest changes in the brain in the formation of brain abscess[27] is usually possible only in subjects who have an early metastatic abscess, but who die of their primary disease while the abscess is still in an early stage. It may rarely be possible also in the case of multiple brain abscesses, in which one of the abscesses is just forming.

The changes which take place in connexion with abscesses of the brain are in principle the same as those associated with abscess elsewhere in the body.

There has been some difference of opinion between various observers in their interpretation of exactly what changes occur in the abscess wall and in the surrounding brain itself. The term "capsule" is a misnomer in most instances, but possibly it may reasonably be applied to the thick fibrous wall formed around a long-standing chronic abscess.

The formation of the so-called abscess wall depends on the virulence of the organism and on the resistance of the infected tissue; the latter may be lowered by a variety of factors—for example, trauma, œdema, and interference with blood supply, which is often bound up with the path of the infection, whether intravascular or perivascular.

At one extreme is the acute onset, usually by the intravascular route, with a highly virulent organism and with little time for the mobilization of the body defences, which produces acute spreading encephalitis. At the

other is the walled off "silent" abscess with no focal symptoms, which develops gradually from a nearby focus of suppuration, possibly with a causal organism of relatively low virulence, but certainly developing slowly enough to allow the brain tissues to mobilize their defences.

Carmichael, Kernohan and Adson[60] have given an excellent account of the evolution of a brain abscess. They have divided their description into the following four phases, chosen because each one represents the advent of a new feature which modifies the existing picture (each stage, of course, merges gradually into the next, and the stages have no particular relationship to the chronological or symptomatic progress of the abscess): Phase I: focal necrosis, microgliosis; Phase II: primary delimitation, fibrosis; Phase III: secondary delimitation, astrogliosis; Phase IV: repair, vascularization.

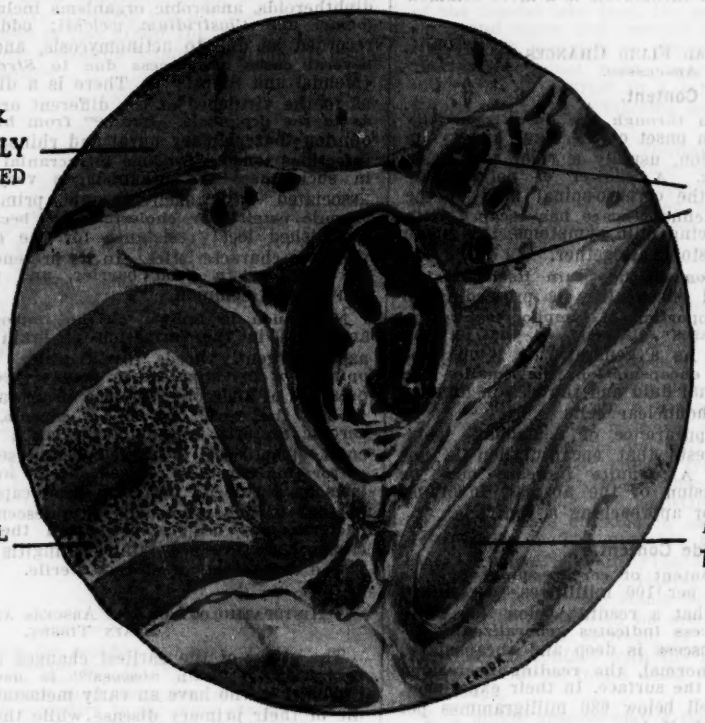

FIGURE VIII.

Veins containing breaking down clot at the entrance to a fissure, very slight perivasculitis and cortical change. (× 200; modified from E. M. Atkinson.[61])

Phase I: Focal Necrosis, Microgliosis.

In the formation of a brain abscess the earliest change is the appearance in the brain of a small localized area which is packed with polymorphonuclear leucocytes, lymphocytes, monocytes, red blood corpuscles and numerous bacteria mingled with disintegrating nervous tissue elements. In the centre of this area a thrombosed blood vessel may be seen, the rupture of which produced this area of early suppuration; or there may be several such areas close together, which are formed by the thrombosis of several tributaries of one larger vessel, and which later fuse. Sometimes there are two or more separated areas with several of these smaller foci of suppuration close together in them, arising from two or three large vascular trunks, the whole fusing when the intervening tissue necroses to form a single large abscess cavity (see Figure XI).

The blood cells have escaped from the lumina of blood vessels near the vessel involved, either by diapedesis or sometimes by rupture.

Invading this necrotic core can be seen *Gitter* cells which are round or oval, usually large and actively phagocytic (see Figure XII). Del Rio-Hortega[62] and his pupils have shown that the microglia, which are mesodermal in origin, are the precursors of the *Gitter* cells, and intermediate irregular, globular, rod and tuberous forms of these cells occur. A number of other workers[63,64,65,66,67] have also confirmed by experiment that the microglial cells participate early in inflammatory reaction of the brain.

As well as hyperæmia, perivascular infiltration and hæmorrhage, occlusive endarteritis and endophlebitis are observed in the vessels. Infiltration of the perivascular spaces of the vessels in the vicinity with all manner of hæmatogenous elements is also a feature. Proliferation of the endothelial cells of the walls of these vessels occurs, and these can be seen wandering out into the tissues. The nuclei of these endothelial cells are often morphologically indistinguishable from nuclei appearing in cells which otherwise have the appearance of *Gitter* cells.[60] This has led to some confusion in description among various observers. Atkinson,[61] for example, maintained that there was very little participation of the microglia at this stage. The proliferation of the endothelial cells is sometimes so great that it leads to absolute occlusion of the vessel. Clumps of endothelial cells arranged concentrically around the capillaries give the whorl-like appearance sometimes seen in sections and described by Atkinson.

The *Gitter* cells are round or oval and usually large and granular, with a vacuolated appearance of their cytoplasm and vesicular nuclei containing usually a prominent nucleolus. By staining with Sudan III the vacuoles in the cytoplasm are shown to be droplets of fat. Oligodendroglia, which Del Rio-Hortega showed to be of ependymal (ectodermal) origin, play an inconspicuous role at all stages of brain abscess formation. At

ILLUSTRATIONS TO THE ARTICLE BY DR. GEORGE SWINBURNE.

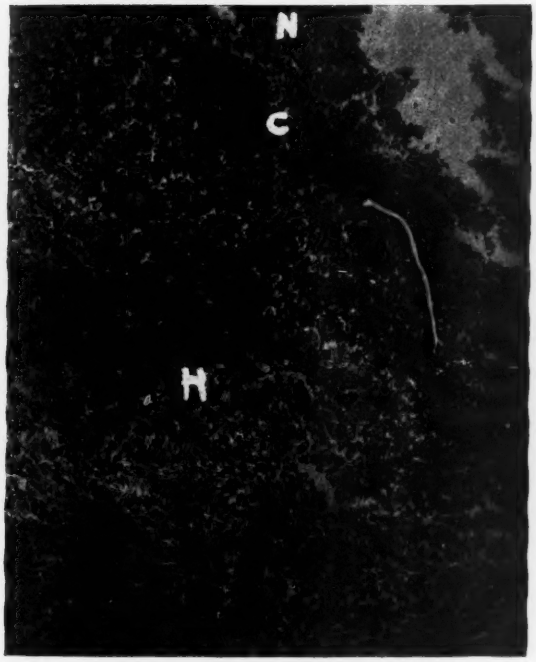

FIGURE XI.

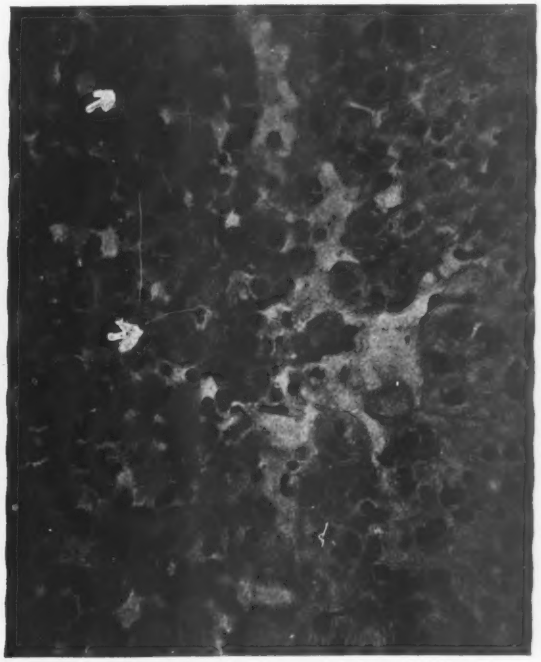

FIGURE XII.

FIGURE XIII.

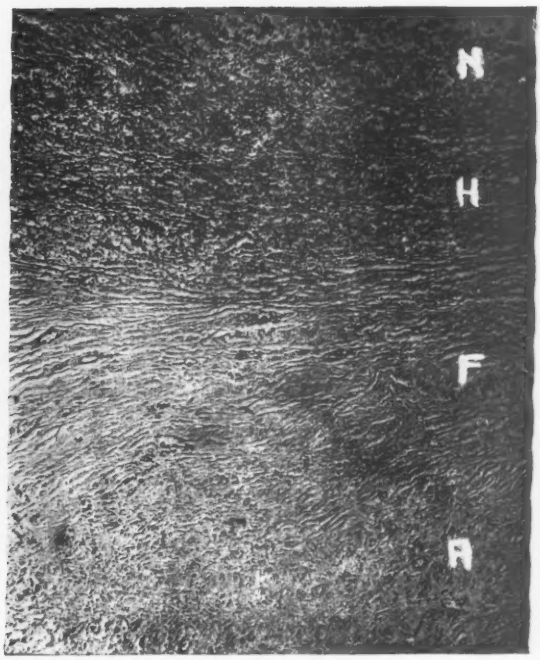

FIGURE XIV.

ILLUSTRATIONS TO THE ARTICLE BY DR. HAROLD W. HORN.

FIGURE I.
Shows the appearance, under the low power of the microscope, of a section through the normal peripheral portion of the placenta.

FIGURE II.
A corresponding section through the tumour, demonstrating strikingly the enormous vascular proliferation and increased cellularity of the interstitial tissue.

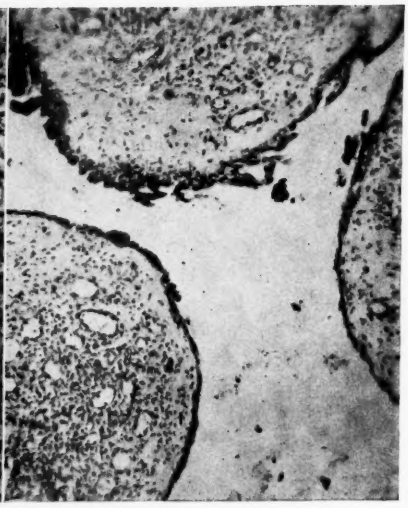

FIGURE III.
Shows the unproliferated trophoblast in adjoining villi in the tumour area.

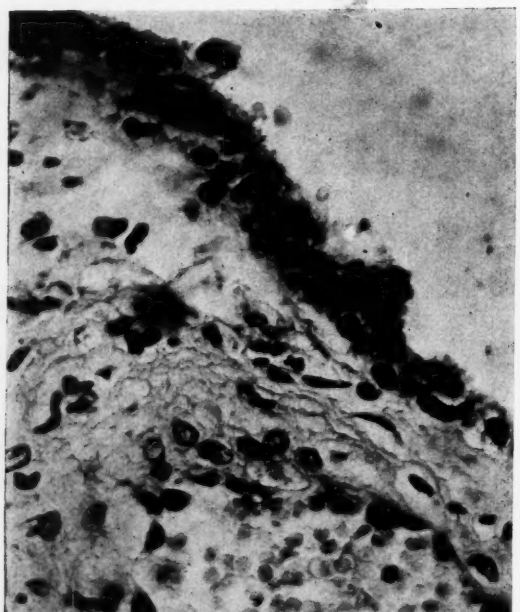

FIGURE IV.
Shows the same under the high power of the microscope, in a single villus.

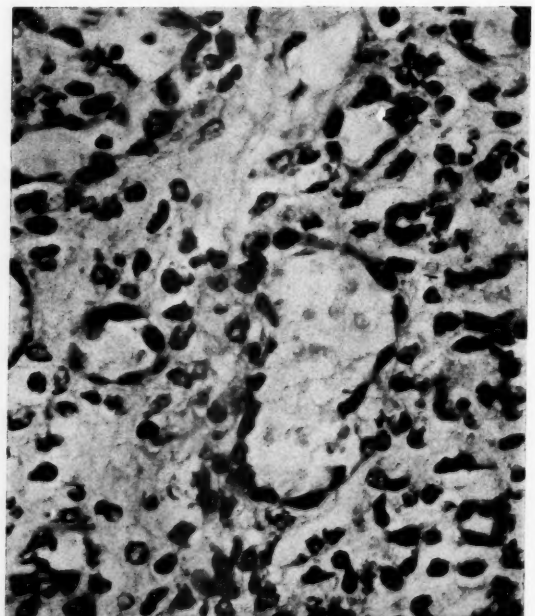

FIGURE V.
The high-power counterpart of Figure II, showing the increased cellularity, together with capillaries, whose walls consist of a single layer of cells, and in whose lumina red cells are present.

this stage the astroglia also play very little part. Nerve cells, if involved in the lesion or in close association with it, show degenerative changes in the form of chromatolysis, pyknosis and cloudy swelling.

Phase II: Primary Delimitation, Fibrosis.

At this stage the central necrotic zone has an amorphous background in which some undamaged polymorphonuclear leucocytes can be seen, but the majority show disintegration in some degree; and here and there are disintegrating erythrocytes and microglia cells.

Endarteritis and endophlebitis of the vessels are not seen in such degree as in the first phase, mainly because the more grossly involved vessels are engulfed in the growing abscess. Hyperæmia and perivasculitis are still pronounced.

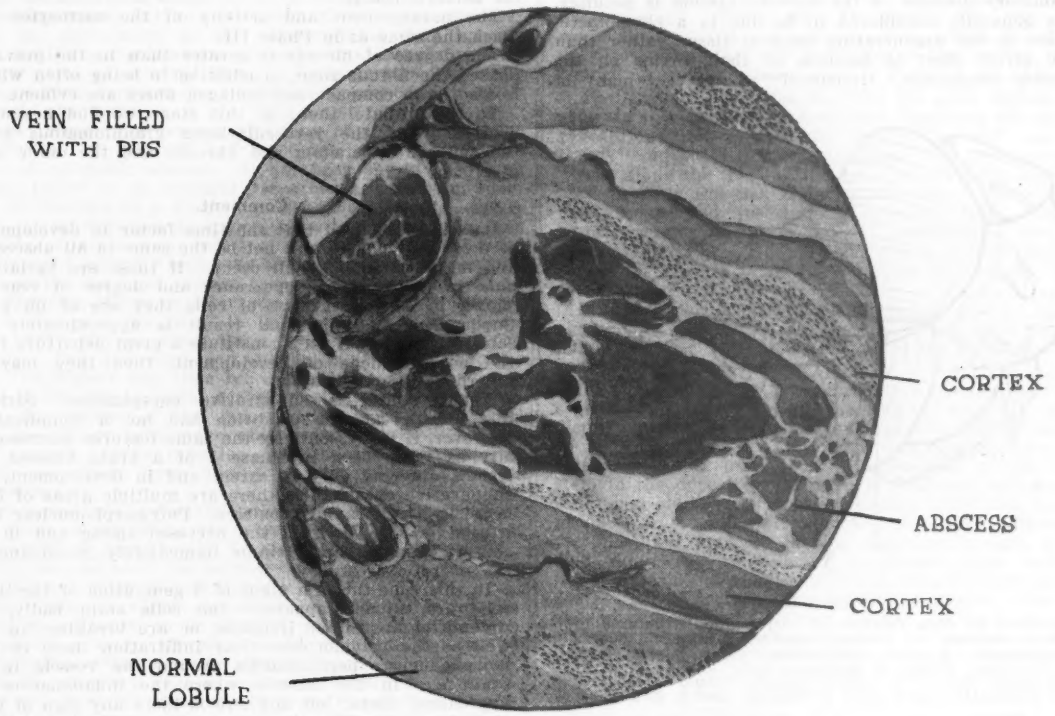

FIGURE IX.

Abscess in centre of a lobule with pial veins containing breaking down clot. (× 200; modified from E. M. Atkinson.[1])

Microglial cells, mainly of the rod and irregularly shaped forms, are seen in the tissue adjacent to the necrotic zone and in lesser numbers at considerable distances in the surrounding brain tissue. Only at the margin of the necrotic zone can much phagocytosis be seen.

The astrocytes are increased in number in the area outside the hyperæmic zone which lies near the necrotic edge. Sometimes in the areas of greatest reaction the *gemästete* forms of Nissl are seen. These are a plump form of astrocyte, in which there is beginning degeneration with swelling and œdema.

Carmichael, Kernohan and Adson think that the giant cells occasionally seen at this stage may be formed by the nuclear division of astrocytes or by the confluence of two or more of these cells.

The nerve cells close to the abscess have disappeared, and further out show degeneration.

In Phase I fibroblasts are rarely seen; but they are seen in Phase II, mainly in the hyperæmic zone close to the necrotic border (see Figure XIII). Their origin has been the subject of controversy. As most of the vessels in this part are capillaries without fixed fibrous tissue cells in their adventitia, it is unlikely that they arise from preexisting fibroblasts, though they certainly do if these are present in the vessel walls. Hassin,[34] from a study of growing membranes, showed that in such circumstances fibroblasts are formed from existing lymphocytes which have migrated into the tissue. Some other observers agree with this, while Carmichael *et alii* point out that nuclei in approximation to existing blood vessels and present in cells which morphologically are already fibroblastic are indistinguishable from adjacent endothelial nuclei. Hence they think that the fibroblasts may be derived from these endothelial cells, and they further observe that, since either postulate is compatible with the fundamental concepts of histogenesis of connective tissue as stated by Maximov,[35] fibroblasts are possibly derived by both means. It is known that if fibrous tissue elements are present in the adventitia of a larger vessel in the neighbourhood, or if portion of the meninges is carried into the cerebral tissue, a greater impetus will be given to fibrosis in the region.

So we have at this stage a central area undergoing liquefaction and necrosis, an adjacent layer of hyperæmia and fibroblastic proliferation, and external to these two zones an ill-defined layer of early astrogliosis.

Phase III: Secondary Delimitation, Astrogliosis.

In the third phase is seen an exaggeration of most of the features of the previous phase. The perivasculitis is now entirely mononuclear. Between the necrotic centre and the zone of hyperæmia and fibroblastic proliferation is now seen a darkly-staining, narrow band, which is composed mainly of fibrin and reticulin, and is the result of the disintegration and condensation of cellular elements and tissue fluids.

The vessels nearest the zone of necrosis show perivasculitis, suggesting that they are not new vessels, but

merely inflamed old vessels (a point enlarged upon later).

Rod and irregular forms of microglial cells are seen in the zone adjacent to the necrotic centre, and *Gitter* cells in among the new fibrous tissue forming in the hyperæmic zone further out.

Just as the appearance and activity of the microglial cells marked the first phase and the appearance of the fibroblasts the second, so the activity of the astrocytes distinguishes the third phase (see Figure XIV). Their activity varies both as to time of appearance and as to extent; they usually begin to become active after the fibroblastic proliferation. In the fibrous zone, the glial fibres freely intermingle with the processes of the fibroblasts and need special staining to distinguish them.

This astrogliosis (commonly referred to as "gliosis", as the oligodendrocytes are inactive in the matter) in inflammatory diseases of the nervous system is peculiar, and is generally considered to be due to a chemotactic influence in the degenerating cerebral tissue rather than to the direct effect of bacteria or their toxins on the astrocytes themselves. Greenfield[30] states that many of

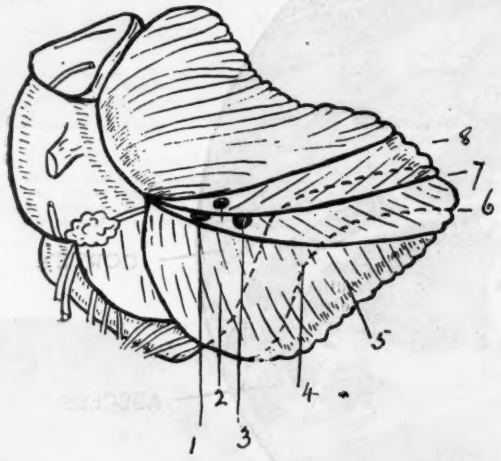

FIGURE X.

Diagram to show relation of surrounding structures to the cerebellum. 1, Internal auditory meatus; 2, *hiatus subarcuatus*; 3, *saccus endolymphaticus*; 4, lateral sinus, 5, biventral lobule; 6, postero-inferior lobule; 7, great horizontal fissure; 8, postero-superior lobule. (Modified by E. M. Atkinson[1] from J. Friesner and A. Braun.[14a])

the lipoids liberated in the disintegration of myelin and of nerve cells possess this power. Cholesterin also, it is thought, may act as a chemical irritant which attracts wandering cells and causes proliferation of fixed cells.

However, this astrogliosis which extends from the inner aspect of the fibrous zone to a considerable distance external to it, is not of very great importance in reinforcing the developing fibrous layer, in which the actual fibrous tissue itself is the dominant factor.

In Phase III there is a great increase in the fibroblasts seen in Phase II, the condition of the nerve cells is much the same, and perivasculitis occurs in vessels right up against the necrotic border.

So that we see in Phase III the central zone of necrosis, then the narrow zone of condensed amorphous cellular debris, then the hyperæmic zone in which fibrous proliferation is occurring, and lastly the outer zone of astrogliosis.

Phase IV: Repair and Revascularization.

As Carmichael *et alii* have pointed out, it is during the fourth phase that the most momentous change in the development of a brain abscess occurs. Where until this juncture the lesion has been advancing and expanding at the expense of cerebral tissue as it were centrifugally, from now on the progress becomes centripetal, being directed toward the liquefied centre in what appears to be an attempt to close the defect.

The narrow, darkly-staining zone of condensed amorphous cellular debris is seen to be invaded and replaced by papillomatous formations containing many newly formed capillary blood vessels. Associated with these is every type of cellular element described in Phase III, except polymorphonuclear leucocytes and nerve cells. Lymphocytes and monocytes have replaced the polymorphonuclear cells.

These newly formed vessels do not appear to have perivascular spaces, the formation of which seems to lag behind the formation of the mesothelial tube of the vessel.

All the types of microglial cells previously described are seen to be taking part in the granulomatous repair of the necrotic centre.

The arrangement and activity of the astrocytes are much the same as in Phase III.

The degree of fibrosis is greater than in the previous phase; the fibrous zone, in addition to being often wider, is also more compact, and collagen fibres are evident.

To recapitulate, then: at this stage we find a central necrotic core, the revascularizing granulomatous zone, the zone of hyperæmia and fibrosis, and the outer zone of gliosis.

Comment.

It will be realized that the time factor in development of these four phases will not be the same in all abscesses, and many variations will occur. If these are variations only in the time of appearance and degree of reaction shown by various groups of cells they are of no great importance, and the final result is approximately the same. However, if they constitute a great departure from the normal mode of development, then they may be regarded as complications.

Among these is suppurative encephalitis. Strictly, this is a preabscess condition and not a complication. However, it has essentially the same features microscopically as those seen in Phase I of a brain abscess, the lesions differing only in extent and in development. In suppurative encephalitis there are multiple areas of focal necrosis which rapidly coalesce. Polymorphonuclear infiltration is seen both in the necrosed tissue and in the acutely inflamed brain tissue immediately in advance of the necrotic area.

In this zone the first signs of degeneration of the tissue cells are already apparent—the cells stain badly, and the nuclei are either irregular or are breaking up. As well as the polymorphonuclear infiltration there is polymorphonuclear perivasculitis around the vessels in the brain and in the fissures where the inflammation has approached them; but nowhere is there any sign of endothelial cell proliferation or grouping of glial cells to form a defensive zone.

This lack of delimitation is due either to a gross imbalance between the virulence of the organism and the resistance of the subject, or to the fact that the patient has died from meningitis or disturbed intracranial hydrodynamics before a defensive reaction has been able to occur. Meningitis is a complication which is probably the commonest single cause of death in cases of brain abscess. On other occasions it takes the form of a localizing adhesive arachnoiditis. Modern chemotherapy has given a considerable measure of control of this complication.

Another complication is diverticulation, or irregular extension of the abscess;[6] unlike suppurative encephalitis it occurs after some degree of delimitation has occurred, or at least after restrictive reaction has begun. Irregularly shaped abscesses instead of the usual spheroid type (in the cerebrum) occur because of this.

Rupture is another complication, and may occur spontaneously, after trauma or after surgical intervention.

Frank hæmorrhage into an abscess is a rare complication. This may be partly due to the endarteritis and endophlebitis of the vessels bordering the necrotic zone, and partly to the fact that the internal pressure in an abscess is greater than the blood pressure in the nearby vessels. As long as hæmorrhage forms an obvious feature

microscopically, an abscess is still advancing and is therefore pathologically young.

With modern chemotherapy and treatment by aspiration, repeated if necessary, it is conceivable that an abscess of the brain may progress to complete healing, and indeed reports of cases are already being published in which this has apparently occurred.

PATHOLOGY IN RELATION TO TREATMENT.

In the approach to a cerebral abscess from the point of view of treatment, the following facts must be considered. The actual cavity of a brain abscess increases in size, because of destruction of brain tissue; but the abscess as a whole increases in size because of the increased number of cells in the area, and because of the œdema, engorgement and hæmorrhage associated with it.

Increase in size by an increase in bulk of tissue means that the additional space has to be obtained in one of the following ways: (*a*) by obliteration of the venous sinuses; (*b*) by obliteration of the cerebro-spinal fluid spaces; (*c*) locally by displacement of the cerebral tissues; (*d*) by compression of cerebral tissue. These factors, especially compression, increase the likelihood of extension of infection in the brain.

The intracranial tension also may be increased by the production of internal hydrocephalus, especially in the case of cerebellar abscess. It has been shown that even slight injury to the cerebral tissue has a depressing effect on its function as a whole. Infected brain tissue, especially if the infection is associated with compression, has a lowered resistance, and a slight injury may produce widespread œdema which may be followed by encephalitis.

Probably the most important point to be considered in the decision when to intervene surgically in a case of brain abscess is the age of the abscess—that is, how many days have elapsed since the infection first invaded the brain. The following facts help in this estimation: (*a*) the history and time relations of the primary infection in the ear or nasal accessory sinuses or elsewhere; (*b*) the course of the pulse and temperature chart; (*c*) the history of a rigor which sometimes accompanies the passage of the infection into the meningeal spaces (this may have been missed by the patient and the attendants).

Pre-operatively, information concerning the condition of the brain surrounding the abscess—that is, the degree of walling-off present—can be gained from a combination of the knowledge of the estimated age of the abscess and the clinical signs and symptoms.

In a case in which the duration of infection of the brain is estimated to be short, a number of focal signs indicating a large abscess will be found to be due to surrounding œdema or sometimes to spreading encephalitis. In a long-standing case a sudden reappearance of symptoms will probably mean that a fresh abscess has formed close to the encapsulated abscess. This is due to an extension of the infection through the wall of the main abscess, and there may be more than one of these secondary abscesses. They may be connected by a fistula with the main abscess, forming a loculated abscess and increasing the difficulties of treatment.

Sometimes the secondary abscess may so increase in size and track around the periphery of the main encapsulated abscess that the latter comes to be free in the former. A localized area of encephalitis with an encapsulated brain abscess lying free in it has been recorded. Perforation into the meningeal spaces is less likely. If an abscess is near the surface of the brain at operation or autopsy, streaks of yellowish, fibrinous-looking material are often seen on each side of the vessels in the sulci immediately over the region of the abscess. Histological examination reveals pus or inflammatory cells in the perivascular spaces, both those in the sulci and those in the white matter around the abscess. Cairns thinks that the leptomeningeal spaces immediately over the abscess are probably sealed off, so that the risk of spreading meningitis after osteoplastic exploration and the passage of a brain needle through an opening in the meninges is lessened. The risk is further minimized by the use of endothermy in sealing off the spaces around the edges of the dural opening.

In an acute abscess, which is little more than a localized area of encephalitis, there is no wall firm enough to cause resistance to a brain needle, although œdematous brain tissue may do so occasionally. The lining of soft, shaggy material is composed of inflammatory cells packed into necrosing brain tissue containing thrombosed blood vessels. Cairns[37] thinks that this class of abscess should not be operated upon or drained or disturbed in any way until it is localized. Supportive and palliative measures and chemotherapy can be employed during the waiting period, and cerebral œdema may be counteracted by means of dehydration, limitation of fluid intake, and the intravenous administration of hypertonic saline solution.

After a few days, the acute symptoms, which are mainly due to the surrounding œdema, will subside. If they do not, for instance in the fulminating cases, in which the encephalitis continues to spread, operation is probably futile, especially with the availability of modern chemotherapy. Those who die during the acute stage could not be saved by early operation, which would serve only to hasten the end and deprive the patient of any chance of arriving at the chronic stage of the abscess.

Another method of dealing with the early abscess is to aspirate it, repeatedly if necessary, observing the strictest precautions not to soil the field. Vincent[38] and also Dandy[39] advocate this. The chances that an abscess will "seed" along the track of the brain needle seem negligible, and certainly the risk is to be preferred to opening into the abscess and inserting a drainage tube, with the added trauma and the risk of encephalitis and post-operative œdema. This method has a greatly increased chance of success with the addition of modern chemotherapy.

Injury to the brain from compression will be guarded against by controlling the cerebro-spinal fluid pressure by appropriate administration of saline solution, or by ventricular or lumbar puncture as indicated.

Brain abscesses may press on and distort the ventricular system, and according to the degree and site of the distortion, may interfere with the pressure of the cerebro-spinal fluid in the various cavities. For example, cerebellar abscesses may cause displacement of the brain stem to the opposite side, with attenuation, and possibly functional obliteration of the aqueduct of Sylvius, and the production of internal hydrocephalus. Withdrawal of cerebro-spinal fluid by lumbar puncture in such a case may cause the medulla to be forced down into the *foramen magnum* with sudden loss of consciousness and death from pressure on the respiratory centre. This may also ultimately occur without lumbar puncture in a case of unsuspected cerebellar abscess, the patient suddenly losing consciousness with little, if any, warning. A ventricular puncture is indicated if such a state of affairs is suspected before operation, either for diagnostic withdrawal of cerebro-spinal fluid or for therapeutic withdrawal to lessen tension. If the abscess is above the tentorium, the lumbar route may be used to withdraw a small quantity of fluid for diagnostic purposes; but even in these circumstances a ventricular "tap" may be wiser for a therapeutic withdrawal.

In temporo-sphenoidal lobe abscess, the anterior horn of the lateral ventricle may occasionally be cut off from the remainder by displaced brain tissue. The withdrawal of from three to six millilitres of cerebro-spinal fluid by means of ventricular puncture from the area in a suspected case will confirm the diagnosis which may have been made by a ventriculogram. One of the reasons why frontal lobe abscesses may become so large and at the same time be well encapsulated is that they do not interfere with the cerebro-spinal fluid circulation because of their forward position. However, perforation of the anterior horn of the lateral ventricle does occur ultimately in progressive frontal lobe abscesses.

An abscess usually does not produce the same degree of pressure that a brain tumour does. This is probably due to the fact that a tumour grows steadily and early interferes with the intracranial fluid balance. In the acute stage of any but a small abscess, œdema superimposed on the local increase in cells, combined with engorgement and hæmorrhage, will produce signs and

symptoms of compression. If the abscess becomes localized there usually follows a period when the actual bulk of the abscess either remains practically stationary or becomes smaller as a result of the subsidence of œdema. False localizing signs may be caused by the œdema of the tissues surrounding an abscess, and temporary paresis due to it will disappear on drainage or aspiration of the abscess with consequent local relief of pressure.

The disturbance of the cerebral function in the majority of cases is due, not to the suppurative nature of the lesion, but to its tumour-like expanding character, and it is this that often forces premature action, and not the need for limitation of suppuration which is being taken care of by the "pyogenic membrane".

The slow pulse rate in cerebral abscess is due to the increased pressure, because when the pressure is relieved by drainage, the rate may rise from 40 to 120 beats per minute. This, of course, also applies to cerebral tumours.

After the acute stage has passed and compression is relieved, the temperature also rises back to normal or a little above it for a few days.

When the abscess has become localized, it may then be drained, or if the surgeon thinks it possible, removed *in toto* like a brain tumour. Chronic thick-walled abscesses are better removed *in toto*.[40] In the treatment of those near the surface, marsupialization has been practised.[41]

There are several reasons why a chronic thick-walled abscess is better dissected out like a tumour. (*a*) Thick-walled abscesses do not always collapse well; consequently there is a risk of pocketing if they are drained by a tube method. (*b*) Even if collapse occurs efficiently as the drainage tube is withdrawn or forced out, the thick fibrous wall remains as a foreign body, and often foci of organisms and pus cells are present in the fibrous tissue; these organisms may lie dormant for twelve to eighteen months or longer, and then may become active and cause a recurrence. (*c*) Removal *in toto* is the best way to deal with loculation.

Abscesses in the form of marginal ulceration, or abscess of the temporo-sphenoidal lobe, either with a "stalk" or just beneath the cortex and just over the tegmen, are probably better drained through the mastoid wound. Due precautions are taken to sterilize the dura and seal off the meningeal spaces, if these are not already naturally sealed off, as in the case of abscesses with a "stalk" or with a fistula into a marginal ulceration. In the treatment of these patients great care must be exercised not to break down any natural defences which have developed. If the abscess is higher in the temporo-sphenoidal lobe or deeper in, exploration and drainage through a separate opening—either trephine or an osteoplastic flap—through a clean field are indicated. The meningeal spaces must be sealed off, preferably with endothermy, and the advantages of removing the abscess if possible *en masse* without spilling its contents, over drainage with the risk of infection of clean brain tissue between the abscess and the surface, are evident.

Cairns and Donald[40] mention seven common errors in treatment: (i) use of the wrong type of operation for the particular case; (ii) missing the abscess with the exploring needle; (iii) the occurrence of blocking of the tube if one of unsuitable type is used; (iv) the use of oblique drainage—that is, an oblique angle from the abscess to the surface of the brain, and obliquely upwards against gravity—this is especially bad; (v) too early interference; (vi) failure to understand the œdema of the surrounding white matter; (vii) errors of nursing, with consequent complications, such as bronchopneumonia.

The ætiology and exact nature of post-operative œdema of the brain are not understood. At post-mortem examination, the white matter around a brain abscess frequently contains an excess of watery fluid between the nerve fibres, sometimes visible to the naked eye, and on occasion, in subjects who have died from the effects of the œdema, the white matter around the abscess is often broken up by collections of this fluid.

The watery or turbid fluid that escapes after the pus, when a brain abscess is drained, is of this nature, exuding from the abscess wall. Cairns and Donald think that post-operative œdema may be wholly or partly caused by spread of infection in the meninges and brain itself at the time the abscess is drained. Spreading meningitis can exist without any of the classical signs of meningitis, and they think that a mild degree of meningitis always occurs after an abscess has been drained. It is not detected because surgeons are loath to perform daily lumbar punctures and cell counts, which in any case are undesirable from the point of view of treatment, since the condition can now be controlled by chemotherapy.

For this reason, Cairns maintains that if the drainage tube has been properly and accurately placed at the first operation, and the symptoms of post-operative œdema arise, it is probably better not to interfere with the tube in the belief that drainage is inadequate, because interference will only increase trauma and hence the spread of infection and œdema. It is better to try to control the œdema with saline therapy, chemotherapy and careful lumbar puncture or ventricular puncture as indicated.

Free dural incision with a rapid change in the pressure balance is likely to be followed by cerebral œdema also.[40]

After evacuation of an acute abscess, the brain fills in the gap within a very short time, and drain tubes are forced out. Collapse of the walls is slower in the older abscesses, depending on the thickness and the degree of fibrosis in the walls.

Usually the dura adheres to the brain over the site of a drained abscess. If the abscess is removed *in toto* and the wound is closed in layers, this does not occur. These adhesions are inclined to cause a sudden jolt to the brain in the vicinity when the patient rises suddenly or otherwise suddenly changes his position in certain directions. If these jolts are repeated over a long period, further fibrotic changes leading to epileptiform seizures may be produced. These seizures usually result either from changes of this nature or from the actual scar of the old abscess, or even from a further scar after removal of one of the above-mentioned causes. Unless it has been very large, or very chronic, it is not usual to find more than a puckering of the brain fibres at the site of an old abscess.

Conclusion.

In conclusion, it may be stated that the whole problem of cerebral abscess, its prophylaxis, treatment and cure, has been profoundly modified by the introduction of modern methods of chemotherapy with drugs of the sulphonamide group and more especially with penicillin. A great many fewer cerebral abscesses are allowed to occur, and the treatment of any that do occur is made easier by combining the particular method of treatment thought appropriate with chemotherapy.

It may also be stated that as yet no finality has been reached on the question of the treatment of cerebral abscess.

The 1939-1945 war provided a greatly increased experience in the treatment of the disease, and doubts have arisen in the minds of some neurosurgeons on several points in the treatment advocated by those who published their views and the results of their work in the immediate pre-war years.

The advisability of the use of drainage tubes at all has been questioned. It is questionable whether it is not better to practise repeated aspiration under chemotherapeutic control, and when the abscess has become stabilized to remove it *in toto*.[44]

In the last ten years the weight of opinion has been in favour of dealing with all temporo-sphenoidal abscesses, with the exception of the marginal ulcerative type, through a separate clean approach and not through the mastoid and ear.

It is only by a correct understanding of the pathology of the lesion that the problem will be finally solved.

Summary.

The surgical pathology of brain abscess, exclusive of tuberculomata and gummata, is discussed under the following headings:

1. A classification of the possible paths of infection.
2. The further development of a formed brain abscess.
3. A clinical classification of the types of brain abscess.

4. The regional pathology of brain abscess occurring in the several lobes of the brain and the cerebellum.
5. The cerebro-spinal fluid changes.
6. Bacteriology.
7. Histopathology of the abscess and surrounding brain.
8. Pathology in relation to treatment.

REFERENCES.

(1) E. M. Atkinson: "Abscess of the Brain", 1934.
(2) A. L. Turner and F. E. Reynolds: "Intracranial Pyogenic Diseases", 1931.
(3) W. MacEwen: "Pyogenic Diseases of the Brain", 1893.
(4) O. Körner: quoted by E. M. Atkinson, *loco citato.*
(5) L. Blau: *"Zur Lehre von den otogenen intrakraniellen Erkrankungen Gehirnabscess-Sinus Thrombose-Meningitis", Beiträge zur Anatomie, Physiologie, Pathologie und Therapie des Ohres,* Volume X, 1918, page 86; quoted by C. B. Courville and J. M. Nielson, *loco citato.*
(6) F. A. Carmichael, junior, J. W. Kernohan and A. W. Adson: "Histopathogenesis of Cerebral Abscess", *Archives of Neurology and Psychiatry,* Volume XLII, 1939, page 1001.
(7) —. Pfeiffer: quoted by Carmichael *et alii, loco citato.*
(8) Lorente de No: quoted by Carmichael *et alii, loco citato.*
(9) —. Cobb: "The Cerebrospinal Blood Vessels", in "Cytology and Cellular Pathology of the Nervous System", by W. Penfield, 1932, Volume II, page 575.
(10) L. H. Weed: "Studies on Cerebrospinal Fluid, II", *Journal of Medical Research,* Volume XXVI, New Series, 1914-1915, page 21.
(11) H. Brunner: quoted by E. M. Atkinson, *loco citato.*
(12) H. Gray: "Anatomy, Descriptive and Applied", 1932, page 761.
(13) W. P. Eagleton: "Intradural Conditions in Relation to Rhinology and Otology", *Archives of Otolaryngology,* Volume XVI, 1932, page 219.
(14) J. Friesner and A. Braun: "Cerebellar Abscess", 1916.
(15) J. S. Fraser: *Journal of Laryngology and Otology,* Volume XLII, 1927, page 839.
(16) F. Miodowski: quoted by E. M. Atkinson, *loco citato.*
(17) E. Urbantschitsch: quoted by E. M. Atkinson, *loco citato.*
(18) J. E. J. King: "Brain Abscess of Traumatic Origin", *The American Journal of Surgery,* Volume XLVII, 1940, page 348.
(19) J. L. Collis: "The Etiology of Cerebral Abscess as a Complication of Thoracic Disease", *The Journal of Thoracic Surgery,* Volume XIII, 1944, page 445.
(20) H. L. Parker: "Metastatic Abscess of the Brain: A Clinical Study", *The American Journal of the Medical Sciences,* Volume CLXXX, 1930, page 699.
(21) C. B. Courville and J. M. Nielson: "Otogenous Abscess of Parietal Lobe, Review of Literature and Report of Six Cases", *Archives of Surgery,* Volume XXX, 1935, page 930.
(22) M. A. Lischoff: "Latent Brain Abscess Secondary to Sphenoid Sinusitis with a Case Report", *Southern Medical Journal,* Volume XXVIII, 1935, page 700.
(23) J. G. Greenfield and F. A. Carmichael: "The Cerebrospinal Fluid", 1925.
(24) C. Bagley, junior: "Brain Abscess due to Gas-forming, Spore-bearing Anaerobes, Report of Two Cases, with Recovery", *Annals of Surgery,* Volume CVII, 1938, page 681.
(25) D. L. Mendel and M. Saibil: "Case of Sub-acute Bacterial Endocarditis with Brain Abscess", *The Canadian Medical Association Journal,* Volume XXXVII, 1937, page 53.
(26) R. Lund: quoted by E. M. Atkinson, *loco citato.*
(27) J. H. Globus and W. L. Horn: "Inherent Healing Properties of Abscess of the Brain, Clinico-anatomic Survey of Fifteen Verified Cases", *Archives of Otolaryngology,* Volume XVI, 1932, page 603.
(28) P. del Rio-Hortega: "Microglia", in "Cytology and Cellular Pathology of the Nervous System", by W. Penfield, 1932, Volume II, page 481.
(29) D. S. Russell: "Intravital Staining of Microglia with Trypan Blue", *The American Journal of Pathology,* Volume V, 1929, page 451.
(30) E. A. Carmichael: "Microglia: An Experimental Study in Rabbits after Intracerebral Injection of Blood", *Journal of Neurology and Psychopathology,* Volume IX, 1929, page 209.
(31) R. B. Wilson: "Brain Repair", *Archives of Neurology and Psychiatry,* Volume XV, 1926, page 75.
(32) C. C. Macklin and M. T. Macklin: "A Study of Brain Repair in the Rat by the Use of Trypan Blue, with Special Reference to the Vital Staining of the Macrophages", *Archives of Neurology and Psychiatry,* Volume III, 1920, page 353.
(33) P. del Rio-Hortega and W. Penfield: "Cerebral Cicatrix: The Reaction of Neuroglia and Microglia to Brain Wounds", *Bulletin of the Johns Hopkins Hospital,* Volume XLI, 1927, page 278.
(34) G. B. Hassin: "Histopathological Studies in Brain Abscess", *Medical Record,* Volume XCIII, 1918, page 91.
(35) A. A. Maximov and W. Bloom: "A Text Book of Histology", 1930, page 48.
(36) J. G. Greenfield: "Inflammatory Cells in the Central Nervous System", in "Cytology and Cellular Pathology of the Nervous System", by W. Penfield, 1932, Volume III, page 1219.
(37) H. Cairns and C. Donald: "The Diagnosis and Treatment of Abscess of the Brain", *Proceedings of the Royal Society of Medicine,* Volume XXVII, 1934, page 1643.
(38) C. Vincent and M. David: "Removal of a Mass of Subacute Abscess of the Cerebral Hemispheres without Drainage", *Revue d'oto-neuro-ophthalmologie,* Volume XIV, 1935, page 1.
(39) W. E. Dandy: "Treatment of Chronic Abscess of the Brain by Tapping: Preliminary Note", *The Journal of the American Medical Association,* Volume LXXXVII, 1926, page 1477.
(40) H. Cairns and C. Donald: "Diagnosis and Treatment of Abscess of the Brain", *The Journal of Laryngology and Otology,* Volume L, 1935, page 73.
(41) J. E. J. King: "Brain Abscess; External Rupture of Capsule with Pericapsular Brain Necrosis", *Annals of Surgery,* Volume CI, 1935, page 190.
(42) J. M. Robinson: "Method of Draining Cerebral Abscesses", *Archives of Otolaryngology,* Volume XXVI, July, 1937, page 49.
(43) B. J. Alpers: "Abscess of the Brain, Relation of the Histologic to the Clinical Features", *Archives of Otolaryngology,* Volume XXIX, 1939, page 199.
(44) R. S. Hooper: Personal communication.

Legends to Illustrations.

FIGURE XI.—Photomicrograph of brain abscess showing (N) necrotic core of inflammatory cells, red blood corpuscles, bacteria and disintegrating nervous tissue, (C) narrow zone of condensed amorphous debris, (H) hyperæmic zone in which fibrous proliferation is occurring. (× 400.)

FIGURE XII.—Photomicrograph of brain abscess showing actively phagocytic *Gitter* cells moving into the zone of condensed amorphous cellular debris adjacent to the necrotic core. (× 600.)

FIGURE XIII.—Photomicrograph of brain abscess showing fibroblastic proliferation in the hyperæmic zone. (× 600.)

FIGURE XIV.—Photomicrograph of brain abscess showing (N) necrotic zone, (H) hyperæmic zone, (F) layer of fibrosis, (A) outer zone. Astrogliosis occurs in this zone as well as in the zone of fibrosis and can be shown by special staining. (× 200.)

Reports of Cases.

CHORIOANGIOFIBROMA.

By HAROLD W. HORN,
Brisbane.

Clinical Record.

MRS. S., aged twenty-six years, a *primigravida,* consulted me on November 21, 1946, her last menstrual period having commenced on September 18. Her due date of confinement was therefore July 2, 1947. Six months previously she had undergone an operation for internal shortening of the round ligaments, with enucleation of three uterine fibroid tumours. She was a thin young woman, complaining of a moderate amount of nausea and vomiting, both of which were relieved by repeated small doses of phenobarbital. Physical examination revealed no gross defects and pelvic measurements were normal. The left nipple was deformed as the result of the removal of a small breast tumour some years before. Her blood group was O, Rh-positive.

Pregnancy proceeded uneventfully until May 17, 1947—that is, until approximately seven and a half months' gestation. On that day her husband reported that she was having hæmorrhage *per vaginam,* with no pain, and she was at once ordered into hospital. I attended her shortly after her arrival, and found that she was then in labour (pains having commenced during transport) and losing bright blood in moderate quantity. Vaginal examination showed that the head was low in the pelvis, the cervix being sufficiently dilated to admit three fingers. Between the head and the rim of the cervix, on the right side, I felt what I considered to be the edge of the placenta—a lateral *placenta prævia.* I was, however, impressed by the unusual thickness of this edge and by its excessive mobility.

As her condition was good, and as the fœtal heart was beating strongly and the rate was normal, I decided to watch her carefully as labour proceeded. In two hours the head was on the perineum, and blood was still trickling from the vagina. At this stage the mother was pale, and her pulse rate was 120 per minute. A small central episiotomy was performed, forceps were applied, and delivery was completed. The cord was immediately tied and divided, and the placenta was delivered at once by Credé's method with ease. Pitocin (one millilitre) was injected intramuscularly.

The child, a male, weighed five pounds eight ounces, and cried weakly. His entire body surface pitted on

pressure. He vomited on various occasions during the next few days, but vomiting and œdema disappeared when feeding was established. The placenta was remarkable, two-thirds of its maternal surface being completely covered by a tumour, which on macroscopic examination was at once accepted as a hydatidiform mole; but it was noted that over a large portion of the tumour the cystic appearance of that condition gave way to red, fleshy projections from its surface. It was undoubtedly the edge of this tumour which presented with the head.

Pathological Investigation.

The placenta was then submitted to Professor A. J. Canny, Professor of Pathology in the University of Queensland, who kindly allowed me to be supplied with sections of the tumour, and with photographs, together with a report which is substantially as follows.

Macroscopic Findings.

The specimen was a placenta which measured 16 centimetres in diameter. The peripheral portion appeared to be normal on naked eye examination. Slightly eccentrically situated was an ovoid area measuring eight by seven centimetres in transverse diameter in which the placental structure was finely polypoid. The polypi ranged from two millimetres to one centimetre in diameter. This polypoid area projected about one centimetre above the level of the uterine surface of the placenta. The umbilical cord appeared normal on macroscopic examination.

Microscopic Findings.

The localized polypoid structure is due to a gross enlargement of the chorionic villi by the growth within their stroma of a fibrohæmangioma of capillary type. Patchy calcification of a few intravascular thrombi and of the trophoblast and decidua has developed, and in some places prolongations of trophoblastic epithelium into the stroma of the enlarged villi show this calcareous change. There is no significant proliferation of the trophoblast. Beyond the limits of the polypoid area there is an abrupt transition to the structure of the normal placenta.

Comment.

Reference to the accompanying photomicrographs (Figures I to V) illustrates clearly the neoplastic changes.

The comparative rarity of tumours of this nature is at once realized when one consults the literature, the only comprehensive report available being an article by Siddall.[1] The "Quarterly Cumulative Index Medicus" also mentions two other authors, but their reports were unavailable. Siddall, in describing his case, reported that in the previous 125 years only 130 placental tumours of angiectatic nature had been recorded, while a few had also been noted in the placentæ of certain animals. He presented the details of one case of his own, making 131 cases in the literature.

The great majority of placental tumours appear as firm elevations beneath the amnion on the fœtal surface; but they have been found on the maternal surface (16 of the above series), and even in succenturiate lobes. Characteristically they are well defined, with a pseudocapsule of compressed and degenerated villi. In one-half of the cases the only connexion with the placenta was by way of blood vessels entering at a hilus, or by a small vascular pedicle. The tumour itself is usually round or oval, but it may consist of many lobes, each facetted and more or less pedunculated. On examination of sections one sees mottled and darkish areas separated by fibrous septa extending inwards from a peripheral fibrous tissue zone; but this appearance is less pronounced in tumours which are predominantly fibrous or myxomatous in structure.

The general structure may be indicated thus:

Tissue	+	Capillaries
↓		↓
fibrous, myxomatous		(i) dilated or collapsed (ii) endothelium—single layer or proliferated

Degeneration, necrosis, calcification or hæmorrhage may occur.

In one-third of the series there was a complete investment of syncytium, or of cytotrophoblast, or of both, in simple or proliferated form. Adjacent placental tissue may be normal, but is usually compressed and shows some degree of degeneration.

The nomenclature has been much debated, but consideration of the foregoing description shows that the term "chorioangiofibroma" is sufficiently comprehensive.

The rarity of these tumours may be more apparent than real, as they vary in size from that of a millet seed to tumours 780 grammes in weight. One might concede that small tumours would frequently escape detection except after meticulous examination.

There seems to be no relationship between the occurrence of these tumours and the age or parity of the mother.

Origin.

The histological findings indicate chorionic origin, but the time of the actual formation of these tumours is not determinable. Pitha, Albert, Beneke and Dienst assume an early origin, at the stage when placental development and vascularization are occurring. Storch described the occurrence of villous hypertrophy in some early abortions and suggested that this represented early chorioangioma.

Clinical Aspects.

The only pathological state encountered in the mother during pregnancy in a series of 110 cases, with greater frequency than in any series of pregnant women, was hydramnios, which was present in 32·7% of this series. There is no special tendency towards toxæmia.

During labour, trouble is met usually only in the third stage, when post-partum hæmorrhage and operative removal of the placenta show increased incidence.

The principal effect of the tumour relates to the fœtus. Premature birth is common, and results from the increased uterine content, hydramnios *et cetera*, or from the stimulating effect of hæmorrhage. Thus there were 41 cases of stillbirth or neonatal death from this cause. The nutrition of the child may suffer, as may be expected from the necessary interference with placental function.

In the cases recorded there was no increase in the mortality rate of full-time babies.

Operative removal of the placenta or tumour may be called for; it occurred six times in the series for each of these indications. Hæmorrhage as an indication arose only four times. In my case, hæmorrhage was encountered in the second and third stages, the child was premature, and malnutrition was present. Subsequent progress was good.

Acknowledgements.

In reporting this case I must acknowledge with thanks the report of Professor A. J. Canny, the interest of Miss Marie Horn in relation to the laboratory investigation, and the excellent review of the subject by Siddall.

Reference.

(1) R. S. Siddall: "Chorioangiofibroma (Chorioangioma)", *American Journal of Obstetrics and Gynecology*, Volume VIII, 1924, page 554; *ibidem*, page 430.

Reviews.

THE PSYCHOLOGY OF ADOLESCENCE.

THE gradual realization that the origin of emotional and mental disorders in adult life generally lies in childhood and adolescence has led to an increasing interest in the problems of those periods, and some aspects of these problems are ably discussed in "The Psychology of the Adolescent" by Leta S. Hollingworth, Ph.D.[1] The book is written ostensibly for parents and teachers, and is free from the psychological jargon so common in similar works.

The problems and conflicts of adolescence are described against the background of the implications of the pubic

[1] "The Psychology of the Adolescent", by Leta S. Hollingworth, Ph.D.; Second Edition; 1947. New York, Toronto and London: Staples Press, Limited. 8½" × 5½", pp. 188. Price: 10s. 6d.

ceremonies of primitive races, and it is shown that they tend to increase the more civilization enforces delay and frustration of such implications.

The author limits the major problems of youth to four groups. Firstly, there is the necessity to leave the family and to become a member of the tribe, rather than of the family group. In the second place, the adolescent is faced with the necessity of making a choice of a vocational career. The third group of problems arise from the fact that the adolescent is physically sexually mature. Fourthly, he is faced with a need for some satisfactory explanation for existence.

The book is mainly devoted to the exposition of these groups. The necessity for psychological weaning by the age of twenty is rightly stressed, but more emphasis might be placed on the dangers to mental health that arise from the absence of such weaning.

An interesting chapter is devoted to the problems surrounding the selection of a vocation.

It appears remarkable that the author has found it necessary to devote pages to proving that whereas a person of high intelligence is intellectually capable of doing low-grade work, one of low intelligence is precluded from adequately filling a position that demands the possession of a high grade of intelligence. One important conclusion may well be quoted here in view of the tendency existing at present to train men in the third and fourth decades for vocations in which they previously showed no interest—often with unfortunate results: "One who has made no beginning in the direction of a satisfying and appropriate occupation by the age of twenty-five is not likely ever to find a good adjustment in this matter."

The chapter on "Mating" is devoted to an objective discussion of the sexual problems of youth; these are sympathetically reviewed by an author who is prepared to recognize the importance of such problems to the adolescent struggling between the Scylla of social disapproval and the Charybdis of ever-present sex stimulation.

In the remaining chapters the author discusses adolescent attitudes to religion and life and to the need that often exists for assistance in finding a satisfactory answer to questions arising therefrom.

This book may be safely recommended to the teacher and parent of the pre-adolescent: "The maximum gain is to be achieved when adolescence is foreseen in the rearing of the child."

HUMAN PHYSIOLOGY.

WINTON AND BAYLISS'S "Human Physiology" is very well produced with good paper, good illustrations and good printing.[1] It has been written by a number of English physiologists, and, perhaps as a consequence of this, the individual parts are first rate, but it is difficult to see any rational basis for the order in which the subjects are presented in the book. For example, the circulatory system is dealt with in the first eighty pages, but the consideration of the properties of cardiac muscle is not presented until page 334. Similarly, the general discussion of the distribution of body water electrolytes and so on occurs much later than the consideration of circulation and blood. For these reasons the student starting off on the book would probably need an instruction sheet to enable him to build up his knowledge from the more general considerations to the particular.

The next feature about the book is that there is a tendency to give information which will be useful clinically; this leads in many cases to a neglect of the simpler underlying principles. When dealing with physico-chemical problems the authors seem to be on somewhat unfamiliar ground; curious notions are introduced, such as electrostatic vacua in connexion with ionic diffusion. The whole chapter on blood-gas exchange suffers from these minor defects, and it is to be hoped that a new edition will be subject to reading by a physical chemist.

Another defect of the book is that no references whatever are given to literature. Physiology is one of the important branches of knowledge in medicine; the medical student finds more and more that he has to go back to original sources for his information. It appears, therefore, to be a mistake not to introduce the student to the wealth of information which occurs in a well-stocked medical library—a text-book should do so. As a consequence of this policy in the book, the authors occasionally mention the name of an individual concerned in the investigation of a particular problem; unfortunately the name selected is not always the most important name.

In being critical of the book in these respects we do not intend to convey the impression that it is not a good book. It is of an ideal size and the content of it is in most respects satisfactory for the pass level in physiology for a medical course. The defects can be overcome by instruction from the lecturer, and in such circumstances the book would serve its purposes very well. It is to be hoped, however, that in future editions the principal authors will exercise a greater integrating activity than they have in the present edition.

[1] "Human Physiology", by F. R. Winton, M.D., D.Sc., and L. E. Bayliss, Ph.D.; Third Edition; 1948. London: J. and A. Churchill, Limited. 9¼" × 6", pp. 608, with many illustrations. Price: 25s.

ENDOTRACHEAL ANÆSTHESIA.

THE second edition of Noel A. Gillespie's "Endotracheal Anæsthesia" comes from the University of Wisconsin Press.[1] The original edition was one of the best contributions to our literature that has so far appeared. The second edition is a revision with some additions, possibly the best of which are the new illustrations. The format remains unchanged. There have been constructive comments relating to the first edition from many people, including some from Australia. These are acknowledged, and the author himself seems most humble about this excellent work.

In the preface to the second edition Dr. Gillespie corrects the misapprehension that the book is meant to cover the full anatomy, physiology, pharmacology, pathology and all theoretical aspects of the subject. He writes: "This work was not written to enable a candidate for examination to acquire rapidly and easily the answers acceptable to an examiner. Its more humble purpose is to be a sound clinical guide to the beginner in the administration of endotracheal anæsthesia; and to provide more experienced workers with a volume which can serve as a convenient and efficient index to the original papers on the subject."

The book is what it claims to be, essentially practical. It is rich in detail useful to a beginner. It is also full of finer points which cannot fail to assist the most expert to improve his practical technique. The chapter dealing with practical applications of endotracheal anæsthesia is particularly valuable, and for this alone the book would be well worth a place on the shelves of any anæsthetist. Although the author claims to have emphasized the practical aspect of this work, each chapter is followed by an extensive bibliography which provides a lead to those desiring to read the subject more widely. Our only regret about such publications as this is that more like it are not available. No one having any aspirations towards anæstaesia should fail to possess and study this excellent book.

THE CARE OF LABORATORY ANIMALS.

THE "Handbook on the Care and Management of Laboratory Animals" prepared by the Universities Federation for Animal Welfare (UFAW) will be gratefully welcomed by those in charge of animal colonies.[2] Laboratories are finding it necessary to devote increased attention to their animals, for if the results of experimentation are to be reliable, it is essential that the breeding stock be carefully selected, that each species be adequately housed and fed according to its particular needs, and that the animals be protected from the diseases to which they are liable.

Detailed information on these points has not been easy to obtain, although notes are given in some text-books and there are articles scattered through a very wide range of journals. The aim of the "UFAW Handbook" is to make generally available the experience gained in animal husbandry in laboratories throughout Britain, and to some extent in other countries. Twenty-five experts were selected to draft the chapters and supplementary information was obtained by means of a questionnaire. The achievement is a credit to "UFAW" and to the editor, Professor A. N. Worden.

[1] "Endotracheal Anæsthesia", by Noel A. Gillespie, D.M., B.Ch., M.A. (Oxon.), M.D. (Wis.), D.A. (R.C.S. England); Second Edition; 1948. Wisconsin: The University of Wisconsin Press. 8¼" × 5½", pp. 268, with many illustrations. Price: $4.00.

[2] "The UFAW Handbook on the Care and Management of Laboratory Animals: With an Appendix on Statistical Analysis", edited by Alastair N. Worden, M.A. (Cantab.), B.Sc. (London), M.R.C.V.S., A.R.I.C., with a foreword by Professor T. Dalling, M.A., M.R.C.V.S., F.R.S.E.; 1947. London: Baillière, Tindall and Cox. 9¼" × 6½", pp. 384, with illustrations. Price: 31s. 6d.

The book begins with an account of the law regulating experiments on animals in Britain. This is followed by an appeal to experimenters to exercise the utmost humaneness in the care and treatment of the animals under their control. Indeed this consideration for the animals' welfare is a thread that runs throughout the book. It is not only ethically imperative, but is also indispensable to experimental success.

There follows a chapter on the general features of an animal house—its construction, temperature control, arrangements for preparation of food, washing and sterilizing of cages, types of cage, provision of water and bedding, methods of identification, and the management of infected animals. Professor Buxton writes on the pests of the animal house and their control.

Stress is laid on personnel. One author regards "choice of the right technical assistant (preferably female) and the training of that right person as by far the most important feature in the management of any rat colony". Again "the adequate supervision of a large mouse colony . . . should be a full-time task for a senior technician of the highest grade. . . . It takes from two to three years to train a good assistant".

The fullest consideration is naturally given to the most commonly used laboratory species—rabbit, guinea-pig, mouse and rat. The chapters on these occupy more than a hundred pages. For each animal there are sections devoted to accommodation, feeding, handling and marking, anæsthesia and euthanasia, and diseases. Full bibliographies are appended.

These four standard animals by no means cover the range of a modern laboratory. For investigations into certain infectious agents, particular species are desirable or essential—ferrets for influenza and distemper, cotton rats for scrub typhus and poliomyelitis, hedgehogs for foot and mouth disease and yellow fever, *et cetera*. The golden hamster is finding increasing use. The South African toad, Xenopus, may be required to meet demands for pregnancy tests. The handbook caters for all these as well as for the wild rat, wild mouse, wood mouse, deer mouse, vole, pigeon, canary, various amphibians and freshwater fish. That so many species need to be discussed illustrates how far animal experimentation has developed since the days when a few guinea-pigs in the basement sufficed.

The book does not include advice on the care of monkeys, dogs, cats, horses and poultry, but references are given to the literature and to experts who are prepared to advise on these animals. Invertebrates receive only casual mention. Brief references are given to inform those who aspire to keep such less usual experimental subjects as vampire bats, crocodiles, shrews, crabs, leeches *et cetera*. No attempt has been made to describe experimental techniques.

It is surprising to find in a book of this nature that seventy pages are occupied by a conspectus of the elements of statistical analysis. The author's intent is to set out the principles and formulæ useful to biologists and to explain them in a way that will be understood by the "intelligent inexpert". He hopes also that a wise planning of experiments will reduce the number of animals that are called on to suffer discomfort or death.

The handbook contains a wealth of practical information, and a copy should be in the library of every laboratory that maintains an animal colony.

AUDIOMETRIC TECHNIQUE.

ALL who have to perform hearing tests will appreciate a useful little guide book such as "Modern Audiometric Technique" by B. M. Green.[1] The author is a practising audiometrist associated with one of the leading hearing aids. The information contained is expressed in simple form and comprises a summary of well-recognized processes of testing of the hearing with a pure tone audiometer and makes some comparison between the use of such an instrument and tuning forks. It would be more complete with a description of the modern processes of testing speech perception with the use of a recorded series of words of accurately controlled loudness. A useful chapter is that on the calculation of percentage hearing loss with the audiometer. Brief mention is made of audiometric diagnosis of the forms of ear disease.

The net proceeds from the sale of the book are to be donated to the Speech Therapy Clinic of the Children's Hospital, Melbourne.

[1] "Modern Audiometric Technique", by B. M. Green; 1947. Melbourne: Modern Hearing Aids Proprietary, Limited. 7¾" × 4¼", pp. 48. Price: 5s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Epilepsy: Psychiatric Aspects of Convulsive Disorders", edited by Paul H. Hoch, M.D., and Robert P. Knight, M.D.; 1948. London: William Heinemann (Medical Books), Limited. 9" × 6", pp. 222. Price: 21s.

The proceedings of the thirty-sixth annual meeting of the American Psychopathological Association, held at New York in May, 1946.

"Introduction to Medical Psychology", by L. Erwin Wexberg, M.D.; 1948. London: William Heinemann (Medical Books), Limited. 8" × 5½", pp. 184. Price: 17s. 6d.

Written by a psychiatrist and based on lectures given to medical students.

"Heart: A Physiologic and Clinical Study of Cardio-Vascular Diseases", by Aldo A. Luisada, M.D., with a foreword by Herrman L. Blumgart; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7", pp. 668, with many illustrations. Price: 75s.

A book intended for the large group of physicians who desire to increase their knowledge of heart diseases.

"Textbook of Gynecology", by Emil Novak, M.D., F.A.C.S.; Third Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 754, with many illustrations, some of them coloured. Price: 60s.

The third edition of a work first published in 1941.

"Bergey's Manual of Determinative Bacteriology", by Robert S. Breed, E. G. D. Murray and A. Parker Hitchens; Sixth Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 1546. Price: £5 12s. 6d.

A book dealing with the classification of bacteria.

"The Skull, Sinuses and Mastoids: A Handbook of Roentgen Diagnosis", by Barton R. Young, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 5¼" × 8", pp. 334. Price: $6.50.

The X-ray appearances and findings on examination of the skull, sinuses and mastoids in health and disease are shown.

"Aids to Organic Chemistry", by Stanley F. Smith, M.B., B.Sc. (London), revised by Ian Leslie, B.Sc., with Introduction by R. H. A. Plimmer, D.Sc., Third Edition; 1948. London: Baillière, Tindall and Cox. 6¼" × 3¾", pp. 140. Price: 4s. 6d.

Intended as a short summary of the essentials of organic chemistry for medical and science students.

"Aids to Pathology", by John O. Oliver, M.B., B.S. (London), M.R.C.S. (England), L.R.C.P. (London); Ninth Edition; 1948. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 342. Price: 13s.

Intended as a concise statement of the subject for the use of medical students.

"Practical Bacteriology, Hematology, and Parasitology", by E. R. Stitt, M.D., Ph.M., Sc.D., LL.D., Paul W. Clough, Sc.D., and Contributors; Tenth Edition; 1948. Toronto: The Blakiston Company. 9" × 6", pp. 1007.

Laboratory procedures are described, interpretation of results is discussed and their correlation with the clinical picture attempted.

"Human Neuroanatomy", by Oliver S. Strong and Adolph Elwyn; Second Edition; 1948. Baltimore: The Williams and Wilkins Company. 10" × 6¼", pp. 456, with illustrations. Price: 45s.

Intended to give the student and practitioner an insight into the structural mechanisms of the central nervous system and their functional and clinical significance.

"Source Book of Orthopædics", by Edgar M. Bick, M.A., F.A.C.S.; Second Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6¼", pp. 554, with few illustrations. Price: 60s.

An attempt to trace the source of orthopædic changes and advances. The first edition has been reorganized—five new chapters and new sections have been added.

"An Index of Treatment", edited by Sir Robert Hutchison, Bt., M.D., LL.D., F.R.C.P., assisted by Reginald Hilton, M.A., M.D., F.R.C.P., and contributors; Thirteenth Edition; 1948. Bristol: John Wright and Sons, Limited. 9¾" × 6¼", pp. 984, with illustrations. Price: 84s.

Intended as a guide to treatment "in moderate compass, and in a form convenient for reference". In this edition over eighty articles are rewritten and all others have been revised.

The Medical Journal of Australia

SATURDAY, AUGUST 14, 1948.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

AN INTERNATIONAL CONFERENCE IN AUSTRALIA ON PNEUMONOKONIOSIS.

At its one-hundred-and-fourth session held at Geneva on March 15 to 20, 1948, the Governing Body of the International Labour Office discussed the holding of an international conference of experts on pneumonokoniosis; it named the year 1949 as the probable date of the conference and Australia as the country in which it would most likely be held. Even at this early date this important event should be noted, and for several reasons. In the first place, pneumonokoniosis is one of the most insidious and crippling of industrial diseases; secondly, a good deal of attention has been paid to it from the aspects of industrial and social medicine, and Australia incidentally has been active in both these spheres; thirdly, the International Labour Office has for some years taken a practical interest in the subject and has devoted two international conferences to it.

Of the insidious nature of silicosis there can be no doubt. In an important discussion on the condition published in this journal in July, 1933, C. G. McDonald laid emphasis on the fact that silicosis sometimes developed very slowly and might produce harmful effects many years after the worker had ceased to be employed in his dangerous occupation. Readers do not need to be reminded of the emphysema often occurring with silicosis or of the frequent superimposition on the damaged lung of a tuberculous infection. Here it is of interest to recall McDonald's statement that he believed that in the absence of extreme emphysema and in the absence of clinical conditions independent of silicosis the majority of silicotic persons worked on until a complicating tuberculosis laid them low. When we think of Australian work on silicosis we call to mind the work of the Technical Commission of Inquiry into the Prevalence of Miners' Phthisis and Pneumonokoniosis in the Metalliferous Mines at Broken Hill (New South Wales). This commission consisted of H. G. Chapman, S. A. Smith, W. A. Edwards, W. A. Sawyer and H. S. H. Wardlaw. (A summary of the commission's report was published in this journal on February 11, 1922.) Much good work has been done at Broken Hill. All men desiring to enter the mining industry must undergo a preliminary radiological and clinical examination in order to establish their physical fitness. If on subsequent examination men are found to be suffering from pneumonokoniosis, either alone or complicated by tuberculosis, they are eligible for compensation. This statement puts the state of affairs baldly—to attempt a full description of the arrangements at Broken Hill and elsewhere in Australia would need a great deal of space and is not necessary—all that is required at the moment is to show that Australia has not been unmindful of the pneumonokoniosis hazard and has taken proper steps to deal with it.

The International Labour Organization has from its earliest days attempted to deal with the pneumonokoniosis problem; the problem was one of the first that it faced. In the account of the proceedings of the one-hundred-and-fourth session of its Governing Body we read that in 1921 the International Secretariat of Stoneworkers adopted resolutions calling for the payment of compensation for silicosis. In 1925, after the adoption of a convention concerning compensation for occupational diseases, the same secretariat asked the International Labour Office to include pneumonokoniosis in the international schedule of occupational diseases for which compensation should be payable. This request was supported by other international unions. After discussion by a special committee of experts a recommendation was made to the International Labour Office to defer the inclusion of silicosis in the list of industrial compensable diseases, because the problem was thought to be particularly difficult and complicated. Subsequently, as a result of further inquiries and of proposals received from South Africa, the first International Conference of Experts on Silicosis was held at Johannesburg in August, 1930. At this conference Australia was one of the eight countries represented. This conference made recommendations as a guide to research under the following six headings: preventive measures; standardization of radiographic technique and terminology; physico-chemical research on dusts; research into the ætiology, pathology and diagnosis of silicosis; the collection and distribution of new information and statistics; bibliography. At its eighteenth session in 1934 the International Labour Conference added silicosis to the list of occupational diseases for which it thought compensation should be payable. After the receipt of requests in 1935 and 1936 from Denmark and Belgium a second International Conference on Silicosis was held at Geneva in September, 1938. Australia was one of nine countries represented and the ten items on the agenda dealt with every aspect of the subject. One of the recommendations of this conference was that similar conferences should be called periodically and it is thought that the time for the calling of such a conference has come. In the report of the March, 1948, meeting of the Governing Body of the International Labour Office it is stated that the medical problem includes: the biochemistry of the pathogenic agent, particularly silica; the process and consequences of accumulation of mineral particles in the lungs; symptomatology; radiography and interpretation of X-ray pictures; diagnosis, including the use of functional tests proposed in recent years and medical and technical preventive measures recently applied. It is also stated that from the medical aspect the methods of examination of workers

and the adoption of a radiological classification of the stages of silicosis are of special importance for medical and medico-legal diagnosis. Reference to the report published in this journal in 1933, which has been mentioned, will show that almost all these aspects were mentioned at the discussion giving rise to the report. A great deal that was undetermined in 1933 still remains to be made clear. It is therefore to be expected that one of the questions to be put before the next conference is "the present state of knowledge of pathogenesis, clinical aspects and diagnosis of silicosis, siderosis, asbestosis, anthracosis and other forms of pneumokoniosis". Another inquiry will be into the present state of preventive measures, that is, the medical and social measures affecting the worker and mechanical and technical measures affecting the workplace. In this inquiry the assistance of physicists, chemists, biologists, engineers and other experts will be needed. The Governing Body refers to the special interest taken in this subject by the Australian Government and this is one of the reasons put forward for the proposal to hold the third International Conference in Australia. The proposal, it should be noted, is not yet a determination. We may take it, however, from the tone of the discussion that Australia is to be the venue of the conference, that the date will be as close as possible to the beginning of 1949, and that the duration will be about a fortnight. The occasion will be of great moment to medicine and to the allied sciences in this country.

Current Comment.

SILICOSIS.

ONE whole number of the *Vierteljahrsschrift der Naturforschenden Gesellschaft in Zürich*, dated December 31, 1947, is devoted to methods of investigating and combating silicosis, which is described as the most important occupational disease in Switzerland from the standpoint of both incidence and gravity. Some fourteen authors, mostly in small collaborative groups, have divided between them the five main chapters of the exposition: (i) "The Position of Silicosis in Switzerland from the Aspect of Workers' Insurance." (ii) "The Results of an Enquiry into Dust in Industries which have Proved to make Workers Prone to this Condition." (iii) "Resting Respiration in Silicosis." (iv) "The X-Ray Picture." (v) "The Combined Physiological-Pathological and Crystallographic and Roentgenographic Investigations into the Silicotic State." A detailed review of the whole work is hardly possible, but certain portions may be taken as typical of the general presentation. In the chapter dealing with respiration we find mention of oxygen absorption and carbon dioxide liberation, respiration frequency, minute volume, vital capacity, complementary and reserve air, dead-space volume and ventilation, alveolar ventilation, oxygen capacity and oxygen saturation of the blood, carbon dioxide content and saturation of the blood, hydrogen ion concentration of the blood and the action of bronchiolar constriction. The section concerned with methods of sampling and analysing dust is excellent, but is confined to those used in the investigation and makes no claim to be comprehensive.

It is in the chapter devoted to the crystallographic and general mineralogical examination of silicotic tissue that the reader will find the most original treatment. Briefly the results are these. In all silicotic tissues investigated quartz has been detected, the only exceptions being indurated centres of long standing where calcification has obscured the silicon dioxide, but even here after removal of the calcium undoubted quartz has been found in some cases. Other minerals of recognized character have also been detected, notably sericite, felspar, magnetite and hæmatite. Felspar and mica are, however, never found alone, but in association with quartz. Crystalline quartz particles have in general an average of 10^{-4} centimetre linear dimension and very rarely less than this. Sometimes the quartz in lymphatic glands exceeds that in the lungs; quartz has also been found in the spleen, and the authors discuss the transfer of the mineral invader from the lungs to the reticulo-endothelial tissues. It is to be hoped that the missing chapters, especially that on bacterial incursion, will soon appear and be treated in the same thorough manner. The X-ray pictures might be a bit clearer in definition. Each chapter has a good bibliography.

THE SURGICAL RELIEF OF ANGINA PECTORIS.

THE pain of *angina pectoris* can cause intense suffering and a degree of inactivity disproportionate to that demanded by the actual cardiac disability. The suggestion that sympathectomy would relieve this pain is said to have been made nearly forty years ago, but not adopted. According to James C. White and Edward F. Bland,[1] this conservative attitude was due in large part to Sir James Mackenzie's dictum that it would be unfortunate if surgeons should discover an operation to relieve the pain of *angina pectoris* because it would remove the vital danger signal which warns the patient when he is overtaxing his heart. White and Bland point out that it has long since been shown that this is not the case, but even today the average practitioner's fears on this score have not been completely dissipated.

The benefits to be gained from the procedure appear to be well supported in the paper by White and Bland, in which they record the experience at the Massachusetts General Hospital with surgical measures for relief of intractable *angina pectoris* during the past twenty years, with a summary of the results obtained in 83 instances. They present a brief history of the procedure, a detailed discussion of the relative neuroanatomy and physiology, and an account of the standard types of surgical interference. Their investigations indicate that all afferent painful impulses from the heart are carried to the paravertebral sympathetic chains either over the middle and inferior cardiac nerves or over accessory cardiac rami which run to the upper three thoracic ganglia. As there are no direct connexions between the cervical ganglia and the spinal cord in the neck, these pathways are all concentrated in the upper part of the thorax, where they reach the highest three or four thoracic spinal nerves over the sympathetic *rami communicantes*. They then enter the spinal cord over the posterior roots. White and Bland suggest that these upper thoracic ganglia, their connecting white *rami communicantes* and the posterior spinal roots are the logical points for attack by the surgeon in the intractable forms of *angina pectoris*. The pathways of cardiac pain may be interrupted effectively by chemical destruction of the sympathetic ganglia and of their rami with alcohol, by sympathetic ganglionectomy or by posterior rhizotomy. It has been found that after these operations there is occasional persistence of mild pain referred to the jaw, the mechanism of which is still unknown. The patients do not lose the danger signal which warns against over exertion, but it is no longer agonizing pain. In the series of 83 cases reported 75 patients had been treated by the paravertebral injection of alcohol and eight by upper thoracic sympathetic ganglionectomy. Of the first group, results described as excellent were obtained in 42 cases, that is to say, the patients were completely or nearly completely relieved of their pain on the side of injection. In 16 cases, intractable *angina pectoris* was so reduced that the patients could be maintained in a state of relative comfort by routine medical measures without the use of narcotic drugs. Six patients failed to obtain adequate relief, five patients were not sufficiently followed up and six died as

[1] *Medicine*, February, 1948.

a direct result of the procedure. It is pointed out that although this mortality rate is nearly equal to that following surgical operation, it is important to bear in mind that this group includes many individuals who would not even have been considered as subjects for surgical interference. Furthermore, the results in the latter third of the series are distinctly better than those obtained earlier because of improvement in injection technique. In the small series of eight cases of upper thoracic ganglionectomy complete relief of pain in the precordium and arm on the denervated side was obtained in all cases. There was one death which, although it occurred a month after operation, the surgeons ascribe to surgery; this death would probably not have occurred if modern chemotherapy had been available at that time. It appears that the open surgical approach gives the most consistently satisfactory relief of pain, but it cannot be used safely for patients with the most advanced forms of coronary disease. Paravertebral block with alcohol, on the other hand, is the safest method, but fails to bring about effective denervation in a small proportion of cases. It is also often followed by intercostal neuralgia, which may be a cause of major complaint, but can be expected to subside within at most three months.

White and Bland recommend that surgical intervention should be considered more often when adequate medical measures fail to control severe and protracted attacks of *angina pectoris*. They assert that in skilled hands surgical intervention is no more dangerous than any of the other common major operations in the corresponding age group. Once relieved of their pain, these patients are able to live happier and more active lives, free from fear of ever-recurring attacks and less conscious of impending death. They are no longer threatened with addiction to morphine, lack of sleep and worry, which so often are factors accelerating termination of life by coronary occlusion. If fatal coronary infarction takes place, it is usually painless. Relief from anginal pain leads to a reduction in the associated nervous tension, more complete relaxation and better sleep, and it seems likely that the patient is thereby given his best chance of surviving a period of critical coronary insufficiency until spontaneous improvement in myocardial circulation occurs by the slow development of competent collateral vessels. No doubt further developments will yet take place in the actual technique of the surgical relief of *angina pectoris*, but with the right indications and if competently performed, the procedure appears to be justifiably well established.

"ROOMING-IN."

Despite the almost universal shortage of hospital beds, it has become more and more popular amongst mothers in most communities to have their babies in hospital. In some cases efforts have been made to provide public nurses to attend mothers in their homes and so to relieve in some measure the strain on hospital accommodation, but generally speaking the advantages to both doctor and patient of childbirth in a satisfactory institution are so obvious that the practice has been encouraged. It has been realized, however, that there are real disadvantages in the usual artificial system in which mother and baby are accommodated quite separately and meet only at meal times. Many mothers have found when the time comes to return home that their babies are comparative strangers and the fathers have sometimes seen their offspring only through a glass panel. This set of circumstances may add quite considerably to the consequent problems of adapting the baby to its home environment. In addition, there has been an interesting swing away from the attitude which tended to regard babies as clockwork machines, a conception which was of course a very convenient one for a precisely run maternity hospital. In an attempt to combine the advantages of hospital care with the more important features of a home environment, the system of "rooming-in" has been devised. It is really rather an obvious solution to the problem and the only major drawback (unfortunately also a very practical one) is the shortage of hospital accommodation facilities. The term "rooming-in" is defined quite simply by Edith B. Jackson,[1] the Associate Clinical Professor of Pediatrics (Psychiatry) at Yale University School of Medicine, as the hospital arrangement whereby a mother may have her baby in a crib beside her bed. At the Grace-New Haven Hospital, where Professor Jackson is an attending pædiatrician, they have formed a rooming-in unit. A unit is housed in the maternity ward solarium which has been partitioned off into a four-bed semi-private ward with attached four-cubicle nursery and doctors' and nurses' office. The baby's crib is usually by the mother's bedside, but may be moved to the nursery at the end of the room as occasion arises. The upper part of each partition is glass with draw curtains, so that there may be visibility or seclusion as desired. Provision is made for ventilation and reduction of noise. The room is cheery and attractive. The rooming-in unit is thus a comfortable division of hospital space wherein four mothers with their newborn babies may be cared for together under unified nursing and medical supervision and where each mother may observe her baby to her heart's content and learn to take care of him before she leaves the hospital. The father is also allowed freer access to his wife and baby.

When the scheme was first proposed, mothers who intended to breast-feed their babies were asked if they would prefer rooming-in to the usual system. Some were not impressed, but the majority favoured the idea, and a later survey revealed that more than half of all expectant mothers (whether they wanted to breast-feed or bottle-feed their babies) were in favour of the idea. The actual basis of selection of mothers for the rooming-in unit has been willingness to nurse the baby, approval of the idea of rooming-in and relative normality. It is interesting to note that married nurses and wives of graduate students, of medical students and of medical staff have been eager applicants for rooming-in and enthusiastic participants. The reaction of both mothers and nurses has been most favourable. This attitude has persisted long after the initial period of novelty might be expected to have passed. An interesting expression of the nurses' reaction is in the rather naïve statement made by the first group of nurses assigned to the rooming-in unit at the end of their two-week assignment: "We know the mothers like rooming-in. We thought you might like to know that the nurses like it too, and when we have babies this is the way we would like to have them."

Many mothers made appreciative comments and were particularly pleased at having the babies under their constant observation and getting to know them. They did not worry about what was happening to them, as all multiparous mothers said they did when their former babies were in the ward nursery. They felt secure in having the same nurse take care of the baby who was taking care of them. They liked being able to ask either the nurse or the doctor questions as soon as questions arose. They learned much in the care of the baby and by watching the nurse during the first few days they gained assurance in taking care of the baby themselves under supervision during the last few days of the hospital period. For the most part, they liked the "ad lib schedule" because they could observe the contentment of the baby. They greatly enjoyed the father's visits and his participation in holding and learning to care for the baby. Multiparous mothers without exception expressed preference for the rooming-in experience over their previous hospital experience. The general experience was that the transition from the hospital to the home was easy. A few mothers did not adapt themselves satisfactorily to the scheme, but even some of these still approved the idea and recommended it to friends and relatives. Follow-up reports by nurses who visited the homes after the mothers left hospital have indicated that the effect on the babies has been favourable. Altogether, this scheme appears to be an eminently sensible and natural arrangement. It may offer difficulties of accommodation and staff, but appears to be well worth encouraging, if at all possible, for the benefit of mothers who desire it.

[1] *American Journal of Public Health and The Nation's Health*, May, 1948.

Abstracts from Medical Literature.

MEDICINE.

Ulcerative Colitis.

M. KIRSHEN (*The American Journal of Digestive Diseases*, December, 1947) states that ulcerative colitis begins in the recto-sigmoid and descending colon and in severe forms spreads throughout the colon and even to the ileum. Colitis may be due to bacillary or amœbic or tuberculous dysentery, *Balantidium coli* or *lymphogranuloma venereum*, or it may be of unknown ætiology. Bergen isolated a diplobacillus as a possible cause. The psychogenic factor is well known. Allergy and relationship to dietetic changes are of uncertain import. Ulcerative colitis is difficult to differentiate from bacillary dysentery proctoscopically. In treatment, rest in bed and a smooth, well-balanced, low residue diet rich in protein, carbohydrates and vitamins are advocated. Transfusions, intravenous administration of glucose and saline solution, vitamins, liver extract, calcium and iron are recommended. Emetine treatment may well be tried. Sulphonamides, penicillin and streptomycin are of doubtful benefit, though sulphonamides should be tried in acute conditions. Vaccines have been successfully used, but the author thinks that they are non-specific in their effects. For diarrhœa, chalk, kaolin, "Kaomagma", "Zymenol", pectin, "Consyl" *et cetera* have a limited value. Morphine is dangerous. "Demerol" in 100 milligramme doses is suggested, and belladonna, papaverine, "Trasentin" and phenobarbital to relieve pain. Psychotherapy including encouragement and optimism are important. Surgery may be necessary, but Bergen and others have reported very unsatisfactory results from 185 ileostomies, 43% of patients operated on dying in six months or less. There should be no operation in the acute or fulminating stage, and ileostomy should always be followed by colectomy; an ileostomy should never be closed.

Recovery of Streptomycin from the Urine.

J. MILLER AND D. ROWLEY (*The Lancet*, March 13, 1948) state that in the treatment of tuberculous patients with streptomycin at least half of the amount of the drug administered is excreted in the urine at a concentration of about 1000 units per millilitre. They describe a simple absorption method whereby from 50% to 60% of the amount of streptomycin in the urine may be recovered in a form suitable for reinjection.

An Artificial Kidney.

N. ALWALL *et alii* (*The Lancet*, January 10, 1948) have constructed an efficient apparatus (Alwall's dialyser) to dialyse a patient's blood outside his body, and they have used it very successfully as an artificial kidney in the treatment of eight persons suffering from uræmia. The patient's blood is made to flow from a radial artery through the apparatus and back into a cubital vein, heparin having been first administered to prevent coagulation of the blood in the dialyser. The dialyser consists of a "Cellophane" tube, about 6500 square centimetres in area, which is wound spirally round a metal or vulcanite cylinder bearing on its outer surface longitudinal grooves, the whole being within an outer cylinder similarly grooved on its inner surface. The space between the two rigid cylinders is so calculated that the "Cellophane" tube can contain only a thin layer of blood of predetermined thickness which cannot be altered. During dialysis, while the blood flows through the "Cellophane" tube, a thin stream of saline solution flows in the opposite direction along the grooves on the cylinders.

Glandular Enlargement in Adults.

H. M. ROYDS JONES (*The Lancet*, December 6, 1947) reports a series of cases in which the condition was clinically similar to glandular fever, but the pathological findings did not support this diagnosis. The author states that the diagnoses of atypical rubella and atypical glandular fever are discounted by the fact that there were no associated typical cases of these diseases. He points out the resemblance of the condition to that described as acute infectious lymphocytosis.

Headache.

S. BUTLER AND W. A. THOMAS (*The Journal of the American Medical Association*, December 13, 1947) discuss the causation and treatment of headache. They say that headaches are produced in four principal ways: distension or dilatation of arteries inside and outside the skull, traction on vessels within the skull, œdema and spasm of muscles of scalp and neck, and pressure on or inflammation of pain-carrying nerves. Migraine and histamine headaches are said to be due to dilatation of blood vessels. However, the early headache of migraine is said to be due to constriction of the internal carotid artery. The second stage of migraine is said to be due to vasodilatation of the external carotid artery, and to be relieved by ergotamine tartrate or dihydro-ergotamine. The third stage of migraine is due to stretching of the scalp with œdema of its vessels and pressure on pain-carrying nerves, and it is not relieved by treatment at the time. The authors state that 85% of migraine is cured or greatly relieved by giving 2·75 milligrammes of histamine acid phosphate in 500 millilitres of isotonic saline solution intravenously on six successive days. Histamine headache occurs paroxysmally several times every day for many weeks. The patients are awakened five or six times a night by severe pain which is relieved when they get up. This headache is also relieved by intravenous injections of histamine. The authors state that many neurologists doubt the existence of histamine headaches of this type. Allergy tests to determine the cause of headache are unreliable in both migraine and histamine headache. Antihistamine therapy with "Benadryl" was ineffective, whereas "Pyribenzamine" was very effective. Tension headaches are due to vasodilatation and are bilateral and occipital (described as a pressure in the back of the neck). Psychic headaches are not headaches; they are strange and terrifying feelings without organic cause. Traction headaches due to traction on the meninges occur after lumbar puncture, and in cerebral tumours. Extracranial headaches are neuralgias or myalgias. Post-traumatic headaches are due to neuritis of the nerves of the scalp involved in injury, anxiety state, adhesions, post-traumatic atrophy of the brain or subdural hæmatoma. The psychogenic factor may be important in all. Toxic headaches of uræmia, eclampsia, jaundice and acute infections are probably due to vascular dilatation or cloudy swelling of the meninges.

Urethane and Leuchæmia.

J. S. HIRSCHBOECK, M. C. F. LINDERT, J. CHASE AND T. L. COLVY (*The Journal of the American Medical Association*, January 19, 1948) describe the effects of urethane in the treatment of leuchæmia and metastatic malignant tumours. Urethane (ethyl carbonate) $H_2NCOOC_2H_5$ is a white crystalline substance which was for a time used as an hypnotic, until it was replaced by the more certain barbiturates. Four subjects of chronic myelogenous leuchæmia had satisfactory remissions after taking one gramme of urethane by mouth thrice daily in capsules. Medication was continued for periods varying between three days and three months. Nausea and vomiting were rare toxic effects. A marked leucopenia was noted during this treatment in many cases of chronic lymphatic leuchæmia, without any apparent benefit to the patient's health. Two patients with Hodgkin's disease did not benefit by the treatment. A patient with giant follicle lymphoma did not improve, and one with lymphosarcoma made a temporary improvement. Eight patients with malignant neoplasms were treated with urethane with little benefit. There were four cases of carcinoma of the stomach with metastases, and one case each of melanosarcoma, carcinoma of the rectum, retroperitoneal myxosarcoma and lymphoepithelioma of the cervical glands, in which the treatment was used; no apparent benefit was observed. In general, although urethane caused a fall in the number of leucocytes, apart from its effect on chronic myelogenous leuchæmia, no benefit was derived from its use.

A New Drug for Myasthenia Gravis.

A. S. V. BURGEN *et alii* (*The Lancet*, April 3, 1948), in a preliminary communication, report the successful use of tetraethyl pyrophosphate, a powerful anticholinesterase, in the treatment of *myasthenia gravis*. The drug is a colourless oil, which may be injected or given by mouth in solution in propylene glycol. Its action lasts about twice as long as that of "Prostigmin".

Respiratory Infections.

J. G. SCADDING (*The Lancet*, January 17, 1948) discusses the pneumonias associated with epidemic respiratory infections. Epidemic respiratory infections may be due to influenza A or B virus, hæmolytic streptococci and psittacosis virus, or there may be some other, at present unknown, cause of these infections. Influenza virus may cause a trivial upper respiratory tract infection or influenzal pneumonia, fatal within a few days, in which there "seems to be" an overwhelming virus infection with simultaneous superinfection by strongly pathogenic bacteria. The author admits, however, that there are many cases of respiratory infection in which the ætiology is complex, or, in other words, not clear.

Discussing primary atypical pneumonia, he states that the name is unfortunate since it implies that other types of pneumonia are typical, and it suggests that the ætiology of primary atypical pneumonia is different from that of other pneumonias. The author suggests that the condition is due to a specifically pneumonotropic virus, because of the absence of the usual bacteria of pneumonia from the sputum. Furthermore he states that the post-mortem appearances show purulent bronchitis and mononuclear exudate in the bronchi, interstitial tissues and consolidated areas. He mentions cold agglutination, but says it is not specific to any one virus or to any particular infection. So-called primary atypical pneumonia varies so greatly in its severity that the author considers it is not due to any one virus. He describes epidemics of pneumonia due to viruses of psittacosis, *lymphogranuloma venereum*, and rickettsia of Q fever. Attempts to isolate a virus from primary atypical pneumonia have met with little success, though Eaton *et alii* appear to have transmitted a virus from atypical pneumonia patients to chick embryos. Endeavours to inoculate human volunteers with nasal washings from patients with primary atypical pneumonia were not conclusive. The author considers the diagnosis unsound.

A New Sign of Coarctation of the Aorta.

S. SUZMAN (*British Heart Journal*, July, 1947) has found that the large collateral arterial circulation around the scapula can be demonstrated in many patients with coarctation of the aorta in whom it is not evident, and that it can be made more obvious in others in whom it is evident, if the patient is made to stoop forward with his arms hanging down vertically while his back is examined in a good light. This is because the greatly dilated subclavian artery is compressed between the clavicle and the first rib; bending forward opens this space and so releases the pressure on the subclavian artery.

Intestinal Carminatives.

S. ALSTEAD AND J. F. PATTERSON (*The Lancet*, March 20, 1948) have assessed the value of different remedies for expediting the passage of flatus from the bowel by the simple method of collecting the gas passed through a rectal tube into a measuring cylinder inverted in water. It was found that carminatives administered orally, hot turpentine stupes applied to the abdomen, radiant heat and the injection of carbachol had no effect in increasing the output of flatus. The intramuscular injection of eight to ten units of pituitrin gave the most satisfactory results, provided that the rectum was empty and a rectal tube was used. There was no advantage found in giving eserine as well as the pituitrin.

The Causes of Clubbing of the Fingers.

E. F. MAUER (*American Heart Journal*, December, 1947) points out that anoxia of the digital tissues is the common factor of the disorders in which clubbing of the fingers occurs. This anoxia may result from arterial anoxia as in some congenital and other forms of heart disease, or it may occur in disorders in which a rapid sedimentation rate of the blood with intravascular formation of rouleaux causes a decrease of the area of corpuscular surface available for diffusion, so that the tissues are insufficiently oxygenated despite a quite high level of arterial oxygen saturation. In all these cases the vascular bed is wide, blood flow is rapid and the hands are warmer than normal. In other diseases, such as rheumatoid arthritis, Raynaud's syndrome and obliterative thromboangiitis, clubbing of the fingers is rare, although tissue anoxia is severe in the latter two and rapid blood sedimentation is characteristic of the first-named. But in all of these blood flow and tissue temperatures are low in the tips of the extremities. It is therefore concluded that for the development of clubbing low oxygen tensions must occur in tissues which are warm and in which the blood flow is greater than normal. These conditions are satisfied when clubbing is present in association with chronic infections, neoplasms and metabolic defects, just as in the classical cases of arterial anoxia.

Myocardial Infarction Treated with Anticoagulants.

HELEN I. GLUECK *et alii* (*American Heart Journal*, February, 1948) have treated each alternate patient out of 88 suffering from acute myocardial infarction with heparin and "Dicumarol". The usual initial dose of "Dicumarol" was 200 milligrammes (unless the prothrombin time was prolonged or the patient was severely shocked). At the same time an intravenous drip injection was started of a 5% solution of glucose in water to which 300 milligrammes per litre of heparin had been added. The heparin was discontinued when the prothrombin concentration fell to 20% to 30% of its normal concentration; this usually occurred in between twenty-four and thirty-six hours, by which time some 300 to 400 milligrammes had been given. The "Dicumarol" was continued for twenty-one days in a dosage regulated by daily prothrombin estimations. The period of twenty-one days was selected because previous work had shown that by the twenty-first day after myocardial infarction the coagulation of the blood was no longer accelerated. There were nine deaths among the 44 patients so treated and three suffered thromboembolic complications; there were 20 deaths among the 44 patients in the control group and thromboembolic complications occurred in 12. The heparin appeared to have a good and sometimes an immediate effect in relieving pain.

Vasospasm Associated with Disseminated Sclerosis.

C. RAY FRANKLIN AND RICHARD M. BRICKNER (*Archives of Neurology and Psychiatry*, August, 1947) examined the fundi and visual fields of eighteen patients with disseminated sclerosis. Different types of constriction of the retinal arterioles were found. These constrictions were usually associated with scotomata which caused blurring of vision or a shimmering appearance when an object was looked at. The constrictions could be removed by vasodilating drugs, as amyl nitrite. The authors suggest that the constrictions were due to spasm and that the nervous lesions of disseminated sclerosis could be due to diminution of the blood flow resulting from this change. The apparent spasm was of two types, transient and permanent, and this accounted for the varying types of symptoms found in disseminated sclerosis. There was no satisfactory explanation of what caused the vasospasm.

Uræmic Œdema of the Lungs.

I. DONIACH (*American Journal of Roentgenology*, November, 1947) states that a proportion of uræmic hypertensive patients with left ventricular failure develop subacute pulmonary œdema which may be transitory. X-ray pictures show massive, centrally distributed shadows in the lungs in spite of minimal local signs and symptoms apart from dyspnœa. In fatal conditions, the lungs show a widespread "solid œdema" due to a fibrinous or an albuminous intraalveolar exudate. This is associated with a mononuclear cell reaction and organization, and hyaline membranes lining the alveolar ducts. The exudate is considered to result from a combination of a rise in pulmonary capillary pressure due to left ventricular failure and an alteration in capillary permeability resulting from uræmia. Though characteristic, the lesion is not specific and is seen in other conditions of heart failure associated with capillary damage, such as rheumatic fever.

Pulmonary Emphysema and Tuberculosis.

ALBERT GUGGENHEIM (*American Journal of Roentgenology*, July, 1947) states that obstructive emphysema may become part of an active progressive primary pulmonary tuberculosis if a caseated lymph node erodes through the wall of a bronchus, thus creating a valve-like mechanism. In many of these cases the emphysema is accompanied by a caseous pneumonic process. If the bronchus is completely occluded, either by caseous material or by extrinsic pressure, an area of atelectasis may develop. So-called epituberculosis is probably identical with such lesions.

Combined Chaulmoograte and Sulphone Treatment of Leprosy and Tuberculosis.

LEONARD ROGERS (*The Lancet*, April 3, 1948) draws attention to the complementary actions of the chaulmoograte and of the sulphones in the treatment of leprosy. The chaulmoograte cause the destruction of vast numbers of the mycobacteria in nodules and other lesions, possibly through some action upon their protective fatty envelopes; but there is some risk that surviving bacilli in softened reacting lesions may be disseminated through the blood and produce new lesions in distant parts. The sulphones exert a very slow action in destroying the microbes in the affected tissues, but they destroy any living ones which may reach the blood stream. Hence they should counteract the risk of harmful reactions resulting from full doses of the more rapidly acting chaulmoograte. A combination of the two drugs, according to the author, is likely to furnish the most rapid and effective treatment of leprosy. The author also suggests that the same principles may be applied to tuberculosis. Colloidal copper morrhuate, which has been used very successfully for two decades in South America, and which produces focal reactions in tuberculosis, might well be worthy of trial in combination with sulphones or with streptomycin.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Rachel Forster Hospital for Women and Children, Redfern, on March 18, 1948, the President, DR. H. R. R. GRIEVE, in the chair. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Leucoplakia of the Cervix Uteri.

DR. IDA SAUNDERS reported a case of leucoplakia of the cervix which had occurred in a patient, aged fifty-one years, who had been married for eleven years, and who had attended for a "check-over" as she thought that it was indicated at her age. Complete examination revealed only one abnormality. The *cervix uteri* was high in the vaginal vault, was twice the normal size and contained one small cyst; on the right side a fibroid polypus half an inch long was attached. There were eight or nine areas of leucoplakia, roughly a quarter of an inch square, on the cervix, and one extended over the pedicle and along half the length of the polypus. Treatment, which consisted in amputation of the cervix, had been difficult because of the size and position of the cervix. The specimen was examined, the fact being recognized that in 2% of such cases malignant changes occurred. The pathologist reported that in the tissue removed from the cervix there was some exfoliation of the squamous epithelium of the cervix with subjacent inflammatory infiltration. The cervical glands were dilated and cystic and in places communicated with the eroded surface. There was no epithelial metaplasia, but there was some keratosis of the surface. The polypoid tissue showed a fibrous stroma containing irregular glands and appeared to have arisen from the cervix, as the margin was covered by squamous epithelium. There was no evidence of malignancy in any of the tissue examined. Dr. Saunders explained that the patient would be seen again in three months; then every six months for the next eight or nine years. Microscopic slides of the pathological material were exhibited.

Cancer Detection Clinic.

Dr. Saunders then discussed the hospital's cancer detection clinic, which was, so far as she knew, the first in Australia. Every patient over the age of twenty-five years was eligible. Patients would be examined *per vaginam* and *per rectum* and the breasts would be palpated. Each would be given an appointment for six months ahead, and the examinations would be repeated indefinitely. Dr. Saunders remarked that the age of twenty-five years might appear to be young, but cancer did come at times in the young. They certainly should urge for the attendance of patients after the age of thirty-five years, but twenty-five years was adopted for the most part in the United States. In Philadelphia, 400 club women had been first investigated and, although very few early cancers were found, many abnormal conditions of the cervix, many fibroids, and some ovarian cysts were detected.

The significance of the examinations lay in the fact that in the breast, ovaries and uterus (body and cervix) cancer was a silent disease for so long, and modern knowledge suggested that the early stages might be longer than was ever imagined. Te Linde, of the Johns Hopkins University, had several cases to support that. His case of macroscopic cancer of the cervix occurring seven years after local removal of supposed non-invasive cancer (sometimes called cancer *in situ*) was well known. Subsequently further sections had been made from the original biopsy, and in one of those, actual invasive cancer was present. He had told Dr. Saunders that he had several other cases in which old pieces of tissue had been reexamined and further sections made. Those showed invasive cancer. Sometimes hundreds of serial sections were made of those cases. Dr. Saunders went on to say that both Ayre at McGill University in Montreal and George Van S. Smith at Boston had shown her examples of simple looking erosion in which biopsy revealed invasive carcinoma, and radical surgery or radium therapy followed the simple treatment which had been undertaken for cure of the "erosion". They had certainly passed the stage at which any erosion should ever be treated without preliminary biopsy. Punch biopsies were easily performed in the surgery.

Dr. Saunders said that she had frequently marvelled at huge cervical growths occurring in married women without post-coital bleeding until a few weeks before examination; and yet at that stage the vagina was almost filled with growth. Surely post-coital spotting must be a very late sign in many cases.

Discussing delay in treatment Dr. Saunders said that figures showed that from ten to twelve months lapsed between occurrence of symptoms and commencement of treatment. A clear mucoid or watery discharge preceded by several months the appearance of blood in many cases of cancer of both body and cervix. The responsibility for that long time was shared by both patient and doctor, largely (Dr. Saunders was speaking as a clinician) by the former, but the doctor was at fault at times.

Dr. Saunders said that they knew that the clinic would receive just criticism because they were selecting some organs and leaving others, but the breast, uterus and ovaries provided women with 65% of all cancers and in addition the rectum would be investigated. The clinic added to the work of the hospital's clerical, pathological and medical staff, and further increased the present very heavy demand upon beds, but it was surely a step in the right direction.

Cancer Cell Cytology.

Dr. Saunders then went on to discuss cancer cell cytology and said that Dr. Spencer, head of the Cancer Research Institution in Washington, felt that just as they now knew of more than one cause of cancer, so they would find more than one cure. He had told Dr. Saunders that Congress had given more money for the study of cancer in 1948 than had ever been granted for the study of a single disease. His feeling was that the study of the cancer cell promised to repay the country well.

Papanicolau, professor of anatomy, had devised the method of staining cancer cells in 1928, but as recently as 1940 he had told Dr. Saunders that New York's big Cancer Hospital had declined to permit him access to its cancer patients. However, early in 1947, they had written asking him to become a consultant there.

Dr. Saunders said that some large hospitals were not engaged in the work at all, and some had been doing it only for two or three years, but their enthusiasm was infectious. Ayre at McGill University was one of the keenest. He had devised a spatula with which he obtained a "surface biopsy" as he called it. It was not a smear and not a biopsy. Papanicolau himself obtained the specimen by suction. The usual vaginal one was universal, but he also had devised a longer one to pass directly into the cervix. It was claimed at Massachusetts that 98% accuracy was given by smears and biopsies combined. Heyman claimed 100% accuracy for biopsies, but Dr. Saunders fancied that he removed larger pieces of the cervix. "False positives" did occur. Interpretation was far from easy. Workers at the Massachusetts General Hospital revised their cases and found erosion in cases in which smears had been taken from the cervical canal. Further experience corrected that. Trout's assistant in San Francisco had told Dr. Saunders of cells from a case of *granuloma inguinale* that could not be distinguished. Papanicolau found some cells in the bronchial secretions of bronchiectasis which gave trouble in diagnosis, although he considered that in investigations of the lung the cancer cell staining was most reliable and most valuable. The position of growths at the periphery and in the upper lobe of the lung was a factor. He stated that they would operate at New York Memorial Hospital for a malignant lung on his smear report in the face of a normal X-ray appearance.

Dr. Saunders said that their work at the Rachel Forster Hospital was in the earliest possible stages; they were learning by taking smears from all subjects of proven cancer who reported there.

(To be continued.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 116, of July 29, 1948.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Volunteer Reserve.

Promotions.—Surgeon Lieutenant Keith Campbell Bradley is promoted to the rank of Surgeon Lieutenant-Commander, dated 27th January, 1948; Acting Surgeon Lieutenant-Commanders Graeme Lindsay Grove and James Stuart Guest, O.B.E., are promoted to the rank of Surgeon Lieutenant-Commander, dated 26th September, 1947, and 27th

January, 1948, respectively; Surgeon Lieutenants Ewen Garth McQueen and Graeme Alvin Robson are promoted to the rank of Surgeon Lieutenant-Commander, dated 31st March, 1947, and 12th August, 1947, respectively.

Royal Australian Naval Reserve.

Promotion.—Surgeon Lieutenant Donald Kerr Grant is promoted to the rank of Surgeon Lieutenant-Commander, dated 8th April, 1948.

AUSTRALIAN MILITARY FORCES.

Interim Army.

Australian Army Medical Corps.

S100029 Lieutenant-Colonel (Temporary Colonel) H. M. Fisher, O.B.E., E.D., relinquishes the appointment of Deputy Director of Medical Services, Headquarters, 4th Military District (Part-Time Duty), the temporary rank of Colonel, is transferred to the Reserve of Officers (Australian Army Medical Corps) (6th Military District) and granted the honorary rank of Colonel, 8th May, 1948.

NX506173 Captain (Temporary Major) M. A. Jackson relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Australian Army Medical Corps) (2nd Military District), 21st May, 1948.

To be Temporary Major, 17th April, 1948.—QX501694 Captain L. R. L. Menogue.

To be Temporary Major, 26th April, 1948.—SX500729 Captain G. G. Wyllie.

Australian Military Forces Wing Repatriation General Hospital (Heidelberg).—SX22303 Captain (Temporary Major) P. R. James relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Australian Army Medical Corps) (4th Military District), 23rd March, 1948.

70th Camp Hospital.—The following officers are transferred to the Reserve of Officers (Australian Army Medical Corps) (2nd Military District): Captains NX208063 E. B. Lee, 19th May, 1948, and NX207264 J. W. Walton, 18th May, 1948.

Reserve Citizen Military Forces.

Australian Army Medical Corps.

5th Military District: To be Honorary Captain, 27th May, 1948.—Maurice Henry Slonim.

6th Military District.—The notification respecting Major F. W. Fay, M.C., which appeared in Executive Minute No. 65 of 1948, promulgated in *Commonwealth Gazette* No. 79 of 1948, is withdrawn.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

Albert Edward Khan (262061) is appointed to a commission with the temporary rank of Squadron Leader, 16th April, 1948, for part-time duties.

Dr. James Augustus Conquest (267263) is reappointed to a commission (part time) with the temporary rank of Squadron Leader, 17th June, 1948.

The appointments of the following Temporary Squadron Leaders are terminated on demobilization: T. Schlicht (257727), 1st June, 1948, R. W. M. Gray (267762), 10th June, 1948.

Reserve: Medical Branch.

The appointment of Temporary Squadron Leader A. E. Khan (262061) is terminated, 15th April, 1948.

Ex-Squadron Leader Theodore Schlicht (257727) is appointed to a commission with the temporary rank of Squadron Leader, 2nd June, 1948.

Correspondence.

RECRUIT MEDICAL OFFICERS FOR THE ROYAL AUSTRALIAN NAVY.

SIR: The Naval Board has advertised and canvassed repeatedly in the last year or more for medical graduates to serve for a short or long period—varying from two to four years or more.

Apparently the young male graduate in Australia is either blatantly self-seeking in his attitude towards medicine, or has forgotten that, although he was unable to serve his country during hostilities, he still has an obligation to fulfil for his salvation by the armed services in that time. For there have been very few applications to join the Royal Australian Navy and none mobilized in this time. The army and air force have also been short of medical officers since the cessation of hostilities.

The present Government of this country, pledged as it is against conscription, has not yet forced post-war graduates to enter the services for a limited period, as has been the case in the United Kingdom, but I earnestly hope that either this power or a subsequent one will see that it is an urgent necessity.

I write as one of a number of wartime reserve medical officers in the Royal Australian Navy who, because of the apathy of the young doctors graduating today, cannot get out of the service to a deserved domestic practice. The Naval Board have declared that they cannot release any more of us until there are recruits to fill our places.

If there are any medical graduates, single and wishing to honour their consciences, and at the same time wishing to avoid giving any government an excuse for "nationalizing" medical services in Australia, let them hasten to contact the Director of Naval Medical Services, Navy Office, Melbourne.

Yours, etc.,

July 30, 1948. "INCENSED."

OTHER WAYS OF DOING THINGS.

SIR: In Dr. Abbie's interesting address (THE MEDICAL JOURNAL OF AUSTRALIA, June 12, 1948) which I have just received in Fiji, he claims that as the picture is reversed on the retina, therefore the infant starts by seeing objects upside down and right to left, and to this he attributes a great deal of the infant's lack of precision in responding to visual stimuli.

This reversal on the retina is a matter of pure optics, and this claim leaves out of account altogether the cerebral factor in vision, yet such reversal is constantly quoted as showing that we start life seeing things topsy-turvy, even in text-books. There is nothing whatever to substantiate this claim or to prove that the child does not see things in correct position from the very start. No doubt the eye may be correctly likened to a camera in that the picture on the retina is reversed, but the camera lacks the next stage present in the human eye, the conduction of the picture by the optic nerve to the brain where the seeing and interpretation is done. The eye equipped with lens has evolved through a vast period of time and a vast number of organisms, and it would be contrary to all expectations that in such a wonderful development the mental interpretation of the optical factors concerned should not have evolved *pari passu* or that the faculty of seeing things as they really are should have shown such a woeful lack of adaptation. The picture is reversed on the retina of all animals equipped with a lens (mammals, birds, reptiles), and if the claim made were true, they would all start life by seeing things topsy-turvy.

In the case of megapodes their eggs are incubated by the heat of sand in deep holes in volcanic areas or on the slopes of active volcanoes, sometimes by the heat of heaps of decaying vegetation, and the young birds on hatching have to fend for themselves. The eggs are very large, so that development is practically complete when the young bird is hatched, fully feathered and with thoroughly efficient wings. The young bird can fly within a few minutes of its hatching, as soon as a slight moisture is dried. I have seen this young bird on its first flight. It is an astonishingly expert business, and it shows no evidence of not possessing perfect orientation.

A young gazelle can run like a hare within a short time of its birth and also shows no sign of not seeing things in their proper position. A calf when approaching its mother for its first drink certainly does not by its movements look as though it saw her stomach and udder upwards.

Surely there is a fallacy in attributing the infant's lack of precision, its vague grasping at objects, to the fact of seeing things upside down. Owing to the early lack of coordinating the movements of the two eyes, it would suffer from a wandering diplopia, its confusion being added to by the consequent periodic absence of stereoscopic vision and the difficulty in judging the distance of objects. As well as this there is the lack of precision in controlling all its muscular movements, due, as Dr. Abbie so interestingly explains, to the slow myelination of the pyramidal tracts.

As Dr. Abbie states: "The human infant is born with less inherent motor capacity than any other animal, and it must learn, often with much labour, such elementary things as how to stand, walk and run, use its hands and talk." And so, in the very occasional case of a child who writes upside down, this can hardly be attributed to vision. Even on the

theory that he starts seeing things reversed, simply because the picture on the retina is reversed, by the age at which he writes, surely this imaginary strange and magical change-over to seeing things normally would have long since occurred. Also, if a child sees, say, a capital A upside down, which I cannot believe, he would, on copying it, not draw it upside down, but correctly, to make it correspond and, to him, also appear upside down. The reason therefore for such writing cannot be visual, but must have some other explanation. I have seen this trait quite often in New Guinea adult natives who have been taught to write in capital letters at an age when it would be quite preposterous to think they did not see things as they are; it often goes with a habit of doing other things the opposite to the usual: placing a pair of boots with the outsides in apposition rather than the insides; putting a box with a hinged lid against the wall with the hinge outwards; lifting a newly packed box and turning it upside down as he shoulders it; placing knives and forks on the opposite sides of a plate to what he has been shown, but not with the points towards you as he would if his action was governed by his retinal picture. The cause must be sought in some peculiar mental inversion, in the realms of psychology, not ophthalmology, or rather, in the argument *re* the child, optics, pure and simple.

Yours, etc.,

Suva, ERIC POCKLEY.
July 16, 1948.

MEDICAL FEES AND NATIONALIZATION OF MEDICINE.

SIR: In your current issue Dr. Molesworth writes of medical fees and the nationalization of medicine. This letter is timely and I think accurate except in one instance. He states "the only increase in fees has taken place in the matter of X-ray work, radiographic or radiotherapeutic. This increase has been forced upon medical practitioners by the vastly increased cost of apparatus and especially of tubes and valves".

The first part of this, with deference to Dr. Molesworth, is not correct. The schedule of diagnostic fees, as issued by the Australian and New Zealand Association of Radiologists, is exactly the same now as in 1931. Even in 1916 the fee for an opaque meal was £5 5s., and it is still the same today. The fee for an examination of the teeth at that time was also £5 5s. It is now £3 3s.

The cost of a first-class mechanically rectified generator in 1930 was about £600. With introduction of valves it rose to £850, and at present a standard generator costs £1250 without accessories. In 1926 the best table and tube stand cost about £450. In 1930 it was about £700 and today it is £1100.

In 1930 the tubes in use cost £45. By 1935 the price of shock-proofed apparatus had risen to about £185, and today it is £240, and modern techniques now call for rotating anodes worth £530 as a necessity.

Room rent, where rooms are available, and all other accessories, including valves, films and screens, have risen in proportion. The embryo radiologist is thus faced with a minimal initial expenditure of £4000 to equip himself. In addition he needs large expensive rooms and continuous secretarial assistance. The grim prospect of recouping such an outlay from an initially small practice and with present high taxation deters all but the stoutest hearts and the longest purses.

Existing radiologists to date have been able to absorb these increases silently without rise of fee, firstly because of the increased volume of work, thus lessening the overhead per patient, and secondly because they have been able to organize their increasing practices by the efficient use of trained assistants to do the large amounts of technical work necessary without any loss of overall diagnostic efficiency. This again costs more and more as time goes on. Such skilled technical assistance is necessarily beyond the financial capacity of most beginners. Thirdly because their present plants are (presumably) bought and paid for.

The time will shortly come, however, when, in the interests of the younger men, it will be necessary to raise radiological fees, for such a process cannot go on indefinitely. Yet in spite of all this, there is a tendency amongst some of our confreres to regard us as rapacious and to press for reduction of the current scale. The plain truth is that we have already absorbed as much of the increased expense as can be reasonably expected. The big established practices can carry on, but for the beginner the prospects on present fees are rather bleak.

In radiotherapy the position is much the same. Fees have been at the same level for many years, whereas all costs have risen to the same extent as in radiodiagnosis. It is really worse, for there has not been the same increase in the volume of work referred.

The problem of the increasing cost of medical care is just as evident to the radiologist as to other members of the profession, and it is his appreciation of this problem that has kept fees where they are to date. We have, as Dr. Molesworth says of the profession in general, "been very lenient in the matter of fees". Like our fellow practitioners we also attend on public hospitals. I would hazard an opinion that the radiologists do two-thirds of the X-ray work in this city in an honorary capacity without fee or recompense.

With the exception of the above point I am heartily in accord with Dr. Molesworth's letter.

Yours, etc.,

137, Macquarie Street, ERIC W. FRECKER.
Sydney,
July 28, 1948.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Film Evening.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following films will be shown at the film evening at 8 p.m. on Friday, August 27, 1948, at the Stawell Memorial Hall of the Royal Australasian College of Physicians, 145, Macquarie Street, Sydney: "Hydrotherapy", "Internal Wiring of Jaw Fractures", "Recreational and Occupational Therapy", "Therapeutic Uses of Heat and Cold: Parts I and II", "Correction of Nasal Deformities", "Penicillin". All members of the profession are invited to attend. Further inquiries should be made by communicating with the Secretary of the Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney. Telephone: B 6980-BW 7483.

Public Health.

DISTRIBUTION OF RADIOACTIVE ISOTOPES.

THE following is published at the request of the Director-General of Health, Commonwealth Department of Health.

In June, 1947, the National Health and Medical Research Council set up a standing committee on radioactive isotopes to formulate procedures regarding the distribution of radioisotopes within Australia. With the development of satisfactory methods of distribution the allocation of radioisotopes in treatment is now being carried out by a therapeutic trials committee in each State.

Applications for radioisotopes for treatment should now be made to the appropriate therapeutic trials committee and should be accompanied by full clinical history of the patient and full particulars regarding the intended use. The quantity of isotope required should be indicated. The following are the chairmen of the various State committees: Dr. C. E. Eddy, Commonwealth X-Ray and Radium Laboratory, University Grounds, Carlton, N.3, Victoria; Dr. H. J. Ham, 135, Macquarie Street, Sydney, New South Wales; Dr. A. G. S. Cooper, Queensland Radium Institute, General Hospital, Brisbane, Queensland; Dr. John Mayo, North Terrace, Adelaide, South Australia; Dr. W. P. Holman, Saint Vincent's Hospital, Launceston, Tasmania; Dr. Alan Nelson, 179B, Saint George's Terrace, Perth, Western Australia.

Radiophosphorus is received at fortnightly intervals from the United States Atomic Energy Commission, and it would be desirable if applications could indicate the approximate date on which the isotope is required. Radiophosphorus is made available free of charge to patients in the public wards of hospitals. For private patients a charge of 27s. 6d. per millicurie at issue in Melbourne (*plus* freight) is made. It would be of assistance if applications were accompanied by the patient's name and a statement as to whether the patient is public or private.

The other radioisotope which has been received in Australia for medical use is radioiodine. However, no continuity of

supply has yet been arranged. It would appear that the quantities of this isotope that will be available in Australia will limit its use to tracer studies and to treatment in hyperthyreoid conditions. It is considered unlikely that radioiodine will be received in sufficently large quantities to treat malignant conditions of the thyreoid.

Radioisotopes are made available from the Atomic Energy Commission on the condition that adequate follow-up reports are submitted at six monthly intervals. Accordingly, it is a condition of issue of radioisotopes in Australia that follow-up reports are made available to the therapeutic trials committees at regular intervals. Follow-up report forms are being prepared and will be issued.

The Commonwealth X-Ray and Radium Laboratory is extending its abstracting service of the literature on the use of radioisotopes in treatment, and such abstracts will be made available on application to the laboratory.

The services of the laboratory are available for assistance in the use of radioisotopes, and application should be made directly to the laboratory or through the therapeutic trials committees. The radiological physicist on the local therapeutic trials committee is available for consultation on techniques and protection in the use of radioisotopes.

The Royal Australasian College of Surgeons.

ROYAL COLLEGE OF SURGEONS OF ENGLAND.

Primary Fellowship Examination.

A PRIMARY EXAMINATION for Fellowship of the Royal College of Surgeons of England will be held in Melbourne, Sydney and Dunedin in February, 1949. Immediately information is received concerning date and place for entries, eligibility for examination, amount of examination fee *et cetera*, a further announcement will be made. In the meantime, prospective candidates are asked immediately to register their names and addresses with the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

Obituary.

EDWARD LLOYD PARRY.

WE are indebted to Dr. Harold Norrie for the following appreciation of the late Dr. Edward Lloyd Parry.

The announcement in *The Sydney Morning Herald* of July 5, 1948, that Dr. E. L. D. Parry had died in Melbourne on July 3, 1948, at the age of sixty-four years conveyed to the general public the news that just another doctor had passed away. To those who knew him, however, it meant much more—it meant that they had lost a friend, not the casual acquaintance type, but the self-effacing "friend indeed" whom one seldom saw, but who was always there when he was wanted and who could be relied upon to respond to the call when it came. His plain humanity gave him that sympathy and understanding which made him beloved of his friends.

Son of the late Dr. Davenport Parry, of Picton, New South Wales, he was the last of a long line (seventeen in all) of medical Parrys, and from them he had inherited that soothing manner and human understanding so characteristic of the old type of family physician, which gave his patients a confidence and peace of mind which often made the difference between success and failure in treatment. He graduated M.B., Ch.M. (Sydney) in December, 1908, was appointed junior resident medical officer at Sydney Hospital in 1909, senior resident medical officer in 1910 and superintendent from 1911 to 1914. In June of the latter year he married Miss Rene Gates and commenced practice in Singleton, New South Wales. When war broke out he enlisted in the first Australian Imperial Force as medical officer and saw service with a field ambulance in Egypt and later as a medical officer in Number 14 Australian General Hospital. On demobilization he took up practice in Moree, where in addition to his medical work he took an interest in civic affairs and was for some years an alderman in the local council. Leaving Moree, he practised at Maitland, where he was a member of the local hospital board and chairman for a term. During his sojourn in Maitland he was instrumental in founding a branch of Rotary in the town and was its first president. Later he practised first at Parramatta and subsequently in Marrickville, until finally, in 1943, failing health forced him to relinquish professional work altogether and live privately at Turramurra. In spite of his ill health, however, he "did his bit" in World War II as medical officer first to the Allied Works Council and later at the Prince of Wales Hospital, Randwick. For many years he lectured and examined for the Saint John Ambulance, in recognition whereof he was admitted as an Officer of the Order of the Hospital of Saint John of Jerusalem.

He is survived by his widow, Mrs. E. Lloyd Parry, of Turramurra, and his daughter.

ERIC GUTTMAN.

THE following appreciation of the late Dr. Eric Guttman has been received from Dr. J. D. Russell.

In the year 1944 in London I asked the advice of Dr. (now Professor) Aubrey Lewis as to how most profitably I might employ a few hours allowed me each week for study for the Diploma in Psychological Medicine. It was thus that I met Eric Guttman at the Emergency Medical Services Hospital, Mill Hill, and for some nine months twice a week worked in his wards under his tutelage. I learnt rapidly to appreciate the happy choice of teacher made for me by Dr. Lewis. Guttman's knowledge of neuropsychiatry was indeed encyclopædic. He was equally at home in research or in the clinic. Though he was essentially a modest man, one learnt gradually of his own training in Berlin and Munich, of his great teachers, and of his own original researches in the widespread fields of pharmacology, neurology and psychiatry. A morning spent with him was a stimulating experience. He obviously enjoyed the role of guide and mentor and would expound on his personal experiences with Mescaline, his research at Saint Hugh's, Oxford, on head injuries, frontal lobe function, vocational selection, with that breadth of vision, catholicity of interest, lucidity, and kindly tolerance for the viewpoint of others that were so characteristic of the man. As I write I have a mental image of him—cigarette ever in mouth, friendly yet ever quizzical, ironical yet withal so kindly, greatly gifted yet so modest. He gave generously of his time to me; nothing was a trouble. I had but to ask and seldom even that.

Life did not deal well with him, yet he never complained. One only learnt intuitively and by inference of his frustrations and disappointments, and sensed the disruptions that had ruined his endeavours on more than one occasion. Yet if ever rarely he was moved to refer casually to his misfortunes, it would be with a laugh and a characteristic shrug of the shoulders. It was therefore with real pleasure that I learnt some two years or so ago that he had been assigned to a position at the Maudsley Hospital that was in some small measure in keeping with his qualities.

We corresponded and I never had any inkling that his health was ever the cause of any anxiety. It was therefore a shock to learn of his untimely death at the age of fifty-two years. It is sadly in keeping with the rest of Eric Guttman's life that, when for the third time by his undaunted courage he was on the point of attaining a position so deservedly merited, he should again be cheated. The fates have indeed been harsh, and Eric Guttman deserved so much better.

THEOPHILUS LINNELL O'REILLY.

WE regret to announce the death of Dr. Theophilus Linnell O'Reilly, which occurred on August 2, 1948, at Killara, New South Wales.

RICHARD DUNCAN DAVEY.

WE regret to announce the death of Dr. Richard Duncan Davey, which occurred on August 3, 1948, at Vaucluse, New South Wales.

JOHN WHYTE GRIEVE.

WE regret to announce the death of Dr. John Whyte Grieve, which occurred on August 4, 1948, at Malvern, Victoria.

CHARLES BROWNLOW PYM.

WE regret to announce the death of Dr. Charles Brownlow Pym, which occurred on August 5, 1948, at Burwood, New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Nolan, Jane Dugmore, M.B., B.S., 1928 (Univ. Sydney), 29, Fairweather Street, Bellevue Hill.
Hodgson, John Hargraves, M.B., B.S., 1947 (Univ. Sydney), Base Hospital, Tamworth.
McCarthy, John Hanshaw, M.B., B.S., 1947 (Univ. Sydney), 175, Macquarie Street, Sydney.
Spooner, Robert Dubois, M.B., B.S., 1947 (Univ. Sydney), 134, West Street, Balgowlah.

THE FEDERAL MEDICAL WAR RELIEF FUND.

THE following contributions to the Federal Medical War Relief Fund have been received:

New South Wales.

D. Wallace, £5.
C. R. R. Huxtable, £2 2s.
R. E. Maffey (second contribution), £1 1s.
Total: £8 3s.

South Australia.

A. R. Magarey, £5.
L. J. Pellew, £5 5s.
H. E. W. Lyons, £1 1s.
Total: £11 6s.
Grand total: £19,679 4s. 6d.

Notice.

S. E. JONES MEMORIAL FUND.

THE following subscriptions have been received for the S. E. Jones Memorial Fund:

	£	s.	d.
Mrs. Olding	100	0	0
New South Wales Public Medical Officers' Association	50	0	0
Dr. C. G. McCarthy	10	10	0
Dr. J. Bostock	5	5	0
Dr. W. S. Dawson	5	5	0
Dr. O. Latham	5	0	0
Total	176	0	0

The Medical Secretary, British Medical Association, New South Wales Branch, has kindly undertaken to receive subscriptions. Cheques should be made payable to "S. E. Jones Memorial Fund" and enclosed in an envelope addressed to S. E. Jones Memorial Fund, c.o. Medical Secretary, British Medical Association, New South Wales Branch, 135, Macquarie Street, Sydney.

Medical Appointments.

Dr. D. K. Grant has been appointed out-patients registrar at the Royal Adelaide Hospital, Adelaide.

Dr. John Gardner McGlashan, Dr. Howard Bruce Cumpston, Dr. Wylie Talbot Gibbs, Dr. Harold Frederick Haselgrove, Dr. Leslie Leonard Nordstrom, Dr. Victor Henry Webster and Dr. Lawrence William Alderman have been appointed quarantine officers, Northern Territory.

Dr. F. E. Welch has been appointed honorary clinical assistant to the Gynæcological Section of the Royal Adelaide Hospital, Adelaide.

Dr. J. C. Yeatman has been appointed honorary clinical assistant to the Medical Section of the Royal Adelaide Hospital, Adelaide.

Dr. Suzanne B. Blackburn, Dr. W. G. Duguid and Dr. N. D. Hicks have been appointed associate medical officers at the Royal Adelaide Hospital, Adelaide.

Diary for the Month.

AUG. 15-21.—Australasian Medical Congress (B.M.A.), Sixth Session, Perth.
AUG. 16.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
AUG. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 18.—Western Australian Branch, B.M.A.: General Meeting.
AUG. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 19.—Victorian Branch, B.M.A.: Executive Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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